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MERVYN ARCHDALL, M.D., Ch.M. (Sydney), F.R.A.C.P., F.R.A.C.S.

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KEY TO DATES AND PAGE NUMBERS.

Number.	Date.	Pages.
1	January 5	1 to 32
2	January 12	33 to 64
3	January 19	65 to 96
4	January 26	97 to 132
5	February 2	133 to 168
6	February 9	169 to 204
7	February 16	205 to 240
8	February 23	241 to 276
9	March 2	277 to 312
10	March 9	313 to 348
11	March 16	349 to 384
12	March 23	385 to 420
13	March 30	421 to 456
14	April 6	457 to 492
15	April 13	493 to 536
16	April 20	537 to 572
17	April 27	573 to 608
18	May 4	609 to 644
19	May 11	645 to 680
20	May 18	681 to 716
21	May 25	717 to 752
22	June 1	753 to 788
23	June 8	789 to 824
24	June 15	825 to 860
25	June 22	861 to 896
26	June 29	897 to 932

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Table of Contents.

The R. H. Fetherston Memorial Lecture.[1]

By ARTHUR M. WILSON,

Honorary Gynæcologist, Women's Hospital, Melbourne.

RICHARD HERBERT FETHERSTON was born in May, 1864, at the Women's Hospital, Melbourne, where his father, Dr. Gerald H. Fetherston, was at that time resident surgeon.

Dr. Gerald Fetherston, after leaving the hospital, commenced practice in High Street, Prahran, and his son Richard lived and worked there or in the close vicinity for the whole of his life. He went to a preparatory school at Toorak and then on to Wesley College. He left Australia at the age of seventeen years to take his medical course in Dublin, attending lectures at Trinity College. He completed the course and obtained his licentiate of the Royal College of Surgeons of Ireland in June, 1884, at the age of twenty years, just two years and eight months after he had started his course. He then went to Edinburgh University, and in 1885 obtained the degrees of bachelor of medicine and bachelor of surgery. He returned to Australia and did some general practice with his father, but was appointed a resident surgeon at the Women's Hospital in 1890, and in the following year he was appointed honorary surgeon in the midwifery department. As his seniority increased, he transferred to the gynæcology department in 1901, and he remained in close association with the hospital until he resigned in 1912 to be the first holder of the newly created position of honorary gynæcologist to the Melbourne Hospital. He held this position until 1924, and he was followed by two accomplished gynæcologists, first by Dr. R. H. Morrison and then by Dr. Roy Chambers. Dr. Fetherston was thus in close association with these two hospitals for a period of thirty-five years, and during this time he held many teaching and examining positions.

On the death of his father Dr. Fetherston succeeded to his general practice in Prahran and continued working there until 1912, when he transferred the practice to Dr. R. N. Wawn. From then onwards he devoted himself to his specialist practice in Collins Street, and he continued working there until the end. He died, as he would have wished, in harness. He had seen some patients in his rooms on June 1, 1943, and had made arrangements to see some on the following day. When it was time for him to be called in the morning it was found that he had passed away peacefully in his sleep at the age of seventy-nine years.

As a medical practitioner he was greatly admired by all his colleagues with whom he came in contact. During his long medical career many advances were made in medicine, but he always moved abreast of the times, and he knew and practised all the details of the most modern treatment. He was a skilful and rapid surgeon and an extremely dextrous obstetrician. As a general practitioner in Prahran he was known far and wide for his kindness and thoughtfulness, particularly to the poorer people, and his name will be long remembered.

In addition to his medical activities, his interests were many and varied. His father had been the Principal Medical Officer of the Militia forces in Victoria, and naturally he followed; he joined up in 1887, immediately after his return from Dublin, and served continuously. At the outbreak of the first World War he held the rank of lieutenant-colonel, and he volunteered for overseas service. He was offered the position of assistant director of medical services, First Australian Division, but as his acceptance would have meant that he superseded two lifelong friends—General Williams and Colonel Charles Ryan—he

[1] Delivered at the University of Melbourne on September 5, 1945.

declined the offer. He remained in Australia and soon found himself Director-General of Medical Services, which position he held until the end of the war, when he retired from the army at his own request. He held the position of Director-General of Medical Services with great distinction, accomplishing wonders with a minimum of staff. In the "Official History of the Australian Army Medical Corps in the War of 1914-1918" it is written that from the start to the finish his record is marked by thoughtfulness and unfailing devotion to duty and by a self-effacement that is most unusual in persons of high rank and station.

> He resolutely declined the honours and rewards which were his by full right and which were offered to him. . . . He left a record of untiring energy and enthusiasm, of absolute impartiality in the difficult task of serving conflicting demands of the A.I.F. and the Australian nation, and of wholehearted devotion to duty in the most invidious task laid upon the shoulders of any Australian officer.

In addition to his medical and army work, he took an active part in the Victorian Branch of the British Medical Association. He was president in 1911 and honorary librarian from 1935 until his death. He was a director of the Australasian Medical Publishing Company, Limited, and of the British Medical Insurance Company in Victoria, and was largely responsible for the formation of these bodies. He was the medical officer of Wesley College, and instituted a system of weighing and measuring the boys long before it was thought of in other colleges.

Even with all these activities he still found time to interest himself in civic affairs. He was a justice of the peace and a town councillor of Prahran, and was for some time a member of the Legislative Assembly of Victoria. He was an energetic health officer for Prahran, and instituted many innovations and reforms. It was characteristic of the man that no matter what work he did, he never sought any publicity or self-advancement.

My own acquaintance with Dr. Fetherston began in 1911, when I became his resident surgeon at the Women's Hospital, as also did my great friend, Roy Chambers. Neither of us had any medical background, and we had more or less strayed into the profession. For some reason or other he took a special personal interest in us both—an interest which never waned—and he was always ready to come to our assistance when we required any help or advice. He always referred to Chambers and myself as his "two boys". During his term of office as Director-General of Medical Services, he made two trips abroad, and I felt honoured when he went out of his way to see me on both occasions. In 1915 I met him on the island of Lemnos and in 1918 on the battlefields of France.

History has a habit of repeating itself. In 1918 we were foreseeing the end of the war, and those of us who had not been in practice before, were starting to worry about the future. I can still remember my feeling of relief when Dr. Fetherston assured me that he would take Chambers and myself under his wing and see that we got good starts on our return. When I came back in 1919 I found that he had arranged for me to go into partnership with Dr. R. N. Wawn, in the practice of which he and his father had so ably laid the foundations. To many of my senior colleagues I owe much, but to no one do I owe more than to Dr. R. H. Fetherston. I am, therefore, deeply sensible of the honour which has been conferred upon me by my being asked to give the first R. H. Fetherston Memorial Lecture, and I have chosen as my subject "The Prevention of Maternal Mortality in Childbirth", as I know it is a subject that was very dear to Dr. Fetherston's heart.

The Prevention of Maternal Mortality.

Owing to the war, the publishing of vital statistics has been in abeyance, the last "Victorian Year Book" being the 1941-1942 issue. In New South Wales the report of the special committee provides interesting statistics almost up to date, and the same observation applies to the statistical surveys of the Women's Hospital, Melbourne. The figures quoted subsequently in this lecture are derived from these sources.

The earliest recorded figures for Victoria, for the decade 1871 to 1880, show the maternal death rate as being 65 per 10,000 births. The figures show a gradual decline until, in the period from 1921 to 1925, it was lowered to 40 per 10,000. For the next nine years there was an alarming rise to 55 per 10,000, and for the next four years (1935 to 1939) a fall again to 47. In 1940 the total was 40 per 10,000 births, and in 1941 a record low figure of 36 per 10,000 births was achieved.

Prior to 1925, deaths from criminal abortion were most unfairly included as maternal deaths in pregnancy and childbirth; but since that date they have been recorded under a separate heading. In 1941, 125 maternal deaths occurred in the State of Victoria, and of these, 47—that is, just over one-third—were the result of criminal abortion. Thus, though the maternal death rate was recorded at 36 per 10,000, if the cases of criminal abortion are excluded, the rate fell to 22·75 per 10,000 births.

The maternal deaths in Victoria in 1941 may be roughly classified as due to the following causes:

Criminal abortion (44 from infection and 3 from other causes)	47
Puerperal infection	23
Toxæmia of pregnancy and puerperium	20
Hæmorrhage of childbirth and puerperium	10
Cæsarean and surgical operations	6
Other causes, including ectopic pregnancy, antepartum hæmorrhage, non-criminal abortion and rare complications	19
	125

I have quoted the causes of maternal deaths for only one year, but investigation of those of the preceding ten years shows that the major causes of death still remain in the same order.

These figures bear a striking similarity to those published by the special committee in New South Wales, which were obtained from inquiry into 511 maternal deaths in the metropolitan area in the years between 1939 and 1943:

Criminal abortion	135
Puerperal infection	95
Toxæmia of pregnancy and puerperium	88
Hæmorrhage of childbirth and puerperium	29
Cæsarean and surgical operations	26
Other causes, including 47 deaths from non-criminal abortion	138
	511

The only serious discrepancy in these two sets of figures was in the deaths from non-criminal abortion. In Victoria, of the 49 deaths from abortion, 47 are recorded as being due to criminal interference. In the New South Wales series 182 deaths are recorded, and only 135 are stated to be due to criminal abortion. This high proportion of "non-criminal" deaths is surprising, as I have always taught that when "natural" abortions occur, although they can be very alarming, fatal results and even serious infections are rare.

A discussion on the prevention of maternal mortality would cover the whole field of obstetrics. I shall, therefore, confine my remarks to the five groups which have been separately classified.

Criminal Abortion.

There has been an alarming rise in the death rate from criminal abortion in Victoria during the last twenty years, as is shown in Table I.

TABLE I.

Period.	Total Maternal Deaths.	Deaths Due to Criminal Abortion.	Proportion of Criminal Abortion Deaths in Total Deaths.
1926 to 1929	198	21	10%
1930 to 1934	160	27	17%
1935 to 1939	139	41	29%
1940	128	50	40%
1941	125	47	38%

The elimination of this cause of death is a moral and ethical rather than a medical problem; but perhaps we could join an an educational crusade and inform the young women (and the young men also) of the risks of criminal abortion, with the reminder that of those who escape a fatal result, many will suffer from sterility and chronic pelvic disorders causing them serious disabilities.

Puerperal Sepsis.

If the deaths from criminal abortion are excluded, puerperal sepsis year by year accounts for one-quarter to one-third of the maternal deaths. With the use of the sulphonamide drugs, penicillin and blood transfusions, the results of our treatment have improved; but it would be infinitely better if by prophylaxis the condition did not occur at all. We all know that a patient during labour, the confinement and the puerperium should be treated with the same meticulous care as one who is undergoing a major surgical operation; but do we act on this principle?

In the cases in which sepsis develops, our death rate could be considerably reduced if the appropriate treatment was instituted at the earliest possible time.

Here I should like to put in a strong plea for the more logical interpretation and a more charitable dispensation of the *Nurses' Registration Act*. The nurse is required to report all cases of puerperal pyrexia in which the temperature reaches 100·4° F. on two occasions between the second and the eighth day. The Nurses' Board is concerned only with the interpretation of the act. It is not concerned with the causation of the pyrexia, whether it is cystitis, pyelitis, mastitis, bronchitis or sepsis. The board may impose such severe and stringent penalties that the hospital routine is reduced to complete chaos, and the hospital may even have to close its doors. The result is that all hospital matrons are terrified of what may happen if they report a case. Consequently they delay the reporting of such a case in the hope that the temperature will subside quickly and so that they may avoid reporting it. If the case happens to be one of puerperal sepsis, the appropriate treatment is often delayed, to the detriment of the patient. How much better it would be if the harassed matron could notify the Board of Health on the second or third day, and if the board, in a spirit of helpful cooperation, would arrange immediately for a vaginal swab to be taken. This would in most cases establish the diagnosis, make possible the isolation of the organism and allow the appropriate treatment to be commenced immediately.

As a matter of interest I should like to call attention to the fact that in these cases our bacteriological findings in Melbourne are different from those in Sydney. In the New South Wales findings on examination of the vaginal swabs, 30% of the cases were due to hæmolytic streptococci, 30% to *Bacillus coli communis*, and 30% to non-hæmolytic streptococci. About 70% of cultures from the blood yielded hæmolytic streptococci. In Melbourne, in a large series of cases, it was found that anaerobic streptococci (which were not even mentioned in the New South Wales findings) played an important part, and it is thought that this organism is largely responsible for the thrombophlebitic type of cases, in which high, swinging temperatures are recorded for weeks on end, and in which the patients frequently di. in the sixth to the eighth week from some pyæmic complication. When this type of infection is established, it is resistant to all varieties of treatment; but I believe that if the diagnosis was established early and the patients were treated with penicillin, the death rate would be considerably lowered. At the Women's Hospital, Melbourne, in the period from July 1944, to June, 1945, there were 3,583 deliveries (this figure covers patients who had attended the hospital during pregnancy and also those admitted as "emergencies"). Of these patients, 196 were suffering from some morbid condition, 160 were considered to be suffering from uterine infection and had vaginal swabs taken on the second and third days, with the following bacteriological findings:

Anaerobic streptococci: 68·4% of patients with morbid conditions (134).
Streptococci (group A): 6·6% of patients with morbid conditions (13).
Non-hæmolytic streptococci: 1·0% of patients with morbid conditions (2).
Staphylococcus aureus: 0·5% of patients with morbid conditions (1).
Bacillus coli: 1·5% of patients with morbid conditions (3).
Unspecified organisms: 3·7% of patients with morbid conditions (7).

There were two deaths, one from perforated duodenal ulcer and one from acute yellow atrophy of the liver. Of the other patients, seven contracted an anaerobic streptococcal septicæmia, but they all recovered within a fortnight. The successful outcome of these cases is entirely due to the early diagnosis and appropriate treatment.

Toxæmia of Pregnancy.

Under the heading "toxæmia of pregnancy", only toxæmias of the nephritic type, with or without eclampsia, will be discussed. At the Women's Hospital, Melbourne, from 1910 to 1937, there were 978 cases of eclampsia with 172 deaths—a mortality rate of 17·6%. In the last decade, 1934 to 1943, the mortality rate was 13·4%.

With efficient treatment in cases of toxæmia not complicated by eclampsia, the mortality rate is not more than 2%. Immediately the patient has a convulsive seizure she moves from a group with 2% mortality rate into a group with a 15% to 20% mortality rate. The word "immediately" is stressed, because among the last 1,000 patients at this hospital, of those who died, 89% died within thirty-six hours of the first seizure.

We cannot prevent a patient from contracting a toxæmia, but we should be able to prevent eclampsia in almost every case. When patients are under careful observation, the so-called fulminant and unexpected eclampsia is uncommon. A rise in the systolic and diastolic blood pressure and a sudden increase in the weight will usually give the obstetrician a warning before albuminuria appears. In addition to the usual routine examinations during pregnancy, the following procedures are suggested: (i) At the first examination a careful record of the systolic and diastolic blood pressure is taken. (ii) In all cases in which the blood pressure is over 140 millimetres of mercury, systolic, and 75 millimetres, diastolic, and in cases in which there is a previous abnormal "kidney" or familial history, the urinary efficiency tests are performed. (iii) The blood pressure is recorded at every subsequent examination. (iv) If the blood pressure rises 10 millimetres or more, or if there is a sudden increase in weight, the patient is placed under close observation.

If the patient does become an eclamptic, what is the best treatment? In my thirty-five years' association with the Women's Hospital, Melbourne, I have watched the pendulum swing from ultra-conservatism to drastic surgical measures, with various intermediate stopping places. For many years I have steered an intermediate course, sizing up each particular case on its merits, or rather demerits. However, more recent results obtained from the intramuscular injection of magnesium sulphate have been most impressive. At a recent meeting of our honorary staff it was decided to standardize the treatment, and all the honorary officers present agreed to follow this routine as far as possible. The treatment for a patient who has had a convulsive seizure, before or after her admission to hospital, is carried out in the following order:

1. A hypodermic injection of one-quarter of a grain of morphine is administered *statim*.

2. A enema is given.

3. Ten mils of a 50% solution of magnesium sulphate are given by intramuscular injection. Injections of two mils are repeated every four hours or if a seizure occurs. The maximum dosage must not exceed 24 mils in 24 hours.

4. *Mistura Sennæ Composita* is given, two drachms being administered every four hours.

5. The membranes are artificially ruptured.

6. Fluid intake is restricted.

7. If the urinary secretion is not satisfactory, the intravenous injection of 25% glucose solution is begun. The metallic electrolytes—for example, sodium chloride—are contraindicated.

The following warnings are given:

1. The magnesium sulphate solution must be prepared by a skilful pharmaceutical chemist.
2. Syringes and needles must be boiled and not merely soaked in spirit.
3. The danger of this treatment is respiratory failure. The knee jerks should be tested before each dose. If they are active, there is no danger; if they are not active, the treatment should be discontinued. The antidote is 10 mils of 10% calcium gluconate solution given intravenously.

So far this treatment has given encouraging results. At the hospital we have had 15 cases with no maternal deaths, and only two patients have had any seizures after the first magnesium sulphate injections.

Owing to the seriousness of this condition and to the absolute impossibility of assessing the value of any particular treatment except by its use in a large number of cases, I would urge strongly that eclampsia be made a notifiable condition.

Post-Partum Hæmorrhage.

Deaths from post-partum hæmorrhage account for about one-sixth of the total. They may be roughly divided into two classes, according to whether they are, or are not, associated with ante-partum hæmorrhage. The truism that the commonest cause of death in ante-partum hæmorrhage is post-partum hæmorrhage still holds good.

In dealing with any case of ante-partum hæmorrhage it is, therefore, necessary to be prepared for possible post-partum hæmorrhage. The patient's blood should be typed and the transfusion service warned. In cases of post-partum hæmorrhage not associated with ante-partum hæmorrhage the maternal deaths should be almost entirely eliminated. Mismanagement of the third stage of labour is by far the commonest cause of post-partum hæmorrhage. There have been no recent great advances on the management of the third stage. The precepts we were taught as students still hold good. Patience is the essence of the contract. It has always amazed me how many obstetricians are prepared to wait two or three hours for the arrival of the baby and are not prepared to wait two or three minutes for the arrival of the placenta. Another warning should be stressed. A manual removal of the placenta, performed with due aseptic precautions and when the patient's pulse rate is under 100 per minute, is not a dangerous procedure. If it is not performed until the patient has become exsanguinated and has a pulse rate over 120 per minute, it becomes dangerous to the patient.

Cæsarean Section.

It is difficult to assess exactly how much Cæsarean section affects the maternal death rate, as an overlapping occurs in the compilation of statistics. A maternal death is classified as due to Cæsarean section if the death occurs immediately after operation. If the patient develops puerperal sepsis and dies a week or so after the operation, the death is almost invariably classified as being due to puerperal sepsis.

In the New South Wales series of 511 maternal deaths, 26 were recorded as being due to Cæsarean section, but another 31 were clasified as due to puerperal sepsis. Thus, of the 511 deaths, 57 (11%) were associated with a Cæsarean section. It is impossible to assess what is happening in Victoria. Of the 78 "non-criminal abortion" deaths, three are recorded as being due to operation, but it is not known how many were included under the heading of puerperal sepsis.

At the Women's Hospital, Melbourne, from July, 1939, to May, 1945, 361 Cæsarean operations were performed, including 102 "repeat operations"; there were 10 maternal deaths, and 63 infants were stillborn or died shortly after delivery.

The figures compare unfavourably with those recorded by C. McIntosh Marshall in his book "Cæsarean Section—Lower Segment Operation" (a book which I can thoroughly recommend to all obstetricians). Marshall reports a series of 246 cases, consecutive and unselected, with no maternal mortality, and with a fœtal mortality rate of 5·7%; unfortunately the neo-natal mortality rate is not recorded. His results are a striking commentary on the necessity of team work, as 238 of the 246 operations were performed at one hospital—the Liverpool Maternity Hospital.

I cannot at this time enter upon a discussion on the Cæsarean operation and the relative values of the three types of operation—classical, lower segment and extra-peritoneal. There are indications and contraindications for all of them. However, I should like to remark, first, that though the operation is comparatively easy to perform, no operation in the realms of surgery is so prone to be followed by serious post-operative complications; secondly, that anyone performing the classical operation in cases of infection or suspected infection is looking for trouble; and thirdly, that the choice of the anæsthetic agent is important.

In view of the association of the Cæsarean operation with so many maternal deaths, I would most strongly urge that Cæsarean section be made a notifiable condition.

What of the Future?

In this brave new world that we are all eagerly awaiting and expecting, can anything be done to solve this problem? Public health is a national concern as much as defence, water supply and sanitation, and in all matters dealing with the public health and the improvement thereof we should, with our special knowledge, have a constructive policy and speak with an authoritative voice. Unfortunately the policy is usually constructed by the politicians and then referred either directly or indirectly to us for our approval and blessing. If we in any way voice our disapproval of any part of the policy, we are instantly labelled as being hostile or at least non-cooperative. How much better it would be if we could have constructive policies on such problems of national health and refer them to the politicians!

In 1939 in New South Wales a special committee appointed by the Department of Public Health commenced an inquiry into the possibility of reduction of maternal mortality. A confidential brochure was sent to all registered medical practitioners in the State, giving full information concerning the practical working of the scheme. As part of this scheme a committee, known as the Special Medical Committee, was appointed by the Department of Public Health to investigate all maternal deaths in the metropolitan area of Sydney, and to ascertain how many of these deaths were preventible and whether any extension of the scheme would be necessary to reduce the maternal death rate.

The report of the committee for the years from 1939 to 1943, inclusive, was published in THE MEDICAL JOURNAL OF AUSTRALIA of December 30, 1944. It is well worth close study. I consider it to be the most valuable contribution to the problem of the reduction of maternal mortality that has yet been published in Australia. In addition to its duties in regard to investigation, this committee provides facilities for obtaining consultants, blood transfusions and bacteriological services. Of the 511 maternal deaths investigated, 182 were due to abortions, criminal or otherwise. Of the remaining 329, 177 were classified as non-preventible and 152 as preventible.

The following are my recommendations on the problem, which may serve as a basis for your consideration and discussion:

1. That there should be immediately appointed in Victoria a special medical committee similar to that operating in New South Wales.

2. That the committee forward an annual report to all registered medical practitioners, and in addition present them with a record of any new advances in diagnosis and treatment.

3. That an educational campaign be conducted to lessen the number of deaths from criminal abortion.

4. That the method of notifying puerperal pyrexia be reconsidered, and that efforts be made to arrange facilities for the early diagnosis and treatment of puerperal sepsis.
5. That eclampsia and Cæsarean section should be made notifiable conditions.
6. That registration of all still-births be uniform, the probable cause of death being given (although this does not come within the actual scope of this lecture).
7. That increased facilities be provided and increased time allotted for the teaching of obstetrics to doctors and nurses, and that post-graduate study for both be encouraged.[1]

AN EPIDEMIC OF POLIOMYELITIS OCCURRING AMONG TROOPS IN THE MIDDLE EAST.

By J. E. CAUGHEY, M.D. (New Zealand), M.R.C.P. (London), F.R.A.C.P.,
Lieutenant-Colonel, New Zealand Medical Corps,

AND

W. M. PORTEOUS, M.D., Ch.B. (New Zealand), M.R.C.P. (London)),
Major, New Zealand Medical Corps.

THE purpose of this article is to place on record the details of an epidemic of 40 cases of poliomyelitis which occurred among New Zealand troops in the Middle East. The epidemic is of interest in that it shows that poliomyelitis may arise and be widely propagated among an adult male population and that it may have the same case incidence and the same clinical and pathological features as epidemics affecting children and adults of both sexes.

The epidemic extended from November, 1940, until July, 1941. Two cases occurred on the north coast of Egypt (32° north latitude) and the remainder in the Cairo area (30° north latitude). Although poliomyelitis is endemic in Egypt, the incidence of the disease among the native Egyptians was not significantly increased in Egypt or in the Cairo area in the 1940-1941 period as compared with the two previous years. The disease is endemic in New Zealand, and comparison is made with New Zealand experience.

EPIDEMIOLOGY.

The epidemic, which comprised 40 cases, lasted for nine months and affected no fewer than 16 different units. Thirty-six patients became ill at one or other of the two New Zealand Expeditionary Force base camps (referred to below as base camps I and II), and two cases occurred at New Zealand general hospitals. These base camps and hospitals were all situated in the environs of Cairo. The remaining two cases occurred on the north coast of Egypt, one at Alexandria and the other in the Western Desert.

Throughout the epidemic cases occurred singly or in small groups, with intervals up to eight weeks during which no fresh cases were reported. The majority of the cases in the first half of the epidemic occurred at base camp I, the majority of those in the second half at base camp II. These camps were separated by a distance of about 12 miles.

The first case occurred in November, 1940, in the Western Desert, and the second four weeks later at base camp II. No connexion was traced between these two cases. Again, four weeks later, cases began to occur at base camp I, where within fourteen days seven cases occurred, six in a single battalion ("X" battalion). At the same time one case occurred at a general hospital and another at Alexandria. In the last-mentioned case the illness began on the seventh day of leave from the Western Desert. (The only other case from the Western Desert had occurred two and a half months previously.) After a further interval of four weeks three more cases occurred at base camp I in three separate units; one of these was a further case from "X" battalion.

In the four months between November 1, 1940, and March 1, 1941, 14 cases had occurred. Ten of these patients became ill at base camp I. An interval of nearly eight weeks now occurred in which no fresh cases were reported (Figure I).

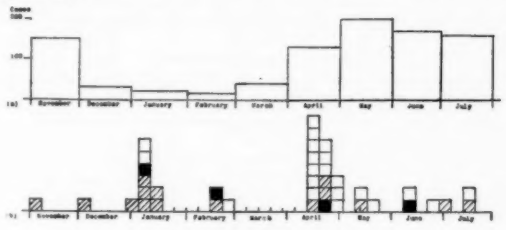

FIGURE I.
Comparison of seasonal incidence of poliomyelitis and diarrhœal disorders among New Zealand troops, from November, 1940, to July, 1941. (*a*) Cases of diarrhœal disorders by months, estimated at a New Zealand general hospital; this does not represent the total incidence of diarrhœa among New Zealand troops, but probably affords a reasonable sample of the whole. (*b*) Cases of poliomyelitis by weeks; each square, one case; the plain squares represent non-paralytic cases, the shaded squares paralytic cases, and the black squares fatal cases.

The second wave of the epidemic began in April, 1941, and 16 cases occurred within two weeks in two adjacent and previously unaffected battalions at base camp II ("Y" and "Z" battalions). Only one case had previously occurred at this camp, three months before. This group of 16 cases was remarkable for its explosive occurrence and for its low incidence of paralysis in comparison with the remaining cases in the epidemic. During this fortnight a single case occurred at a general hospital. The patient was a member of the unit who had not been in contact in the hospital with patients suffering from poliomyelitis. The remaining nine cases of the epidemic occurred at intervals until July, 1941, when the last case was reported. Four of these nine came from three different units at base camp I, and included two more cases from "X" battalion; the other five came from four different units at base camp II.

SPREAD OF INFECTION.

The infection was widespread throughout the New Zealand forces in Egypt, and the long intervals (up to eight weeks) during which no fresh cases arose possibly indicate the existence of a considerable body of immune carriers. The source of infection was not clear in every case; but at each of the two base camps there was a relatively heavily infected "reservoir" from which neighbouring units could have become infected. At base camp I, nine cases occurred in "X" battalion between January 1 and May 16, 1941. Cases were reported from this camp from January 1 to June 25, 1941. At base camp II, "Y" and "Z" battalions, which occupied adjacent lines, provided 16 cases in two weeks (April 18 to May 2). No further cases occurred in these two battalions; but cases were reported from other units at this camp until July 18. Moreover, "Y" battalion was the training unit for an infantry group of which "X" battalion was a part, and interchanges of personnel between the two battalions occurred frequently. In this way infection may have spread from base camp I to base camp II in April. (Before "Y" and "Z" battalions became infected, only one case had occurred at base camp II, three months previously.)

CASE INCIDENCE.

The numerical strength of the New Zealand Expeditionary Force was steadily increasing during the epidemic months, the average for the whole period being approximately 18,000 men. Reckoned on the basis of this figure, the incidence was 2·2 cases per 1,000 of population.

[1] Since this lecture was given, 13 additional patients with eclampsia have been treated with injections of magnesium sulphate, making a total of 28. Of these only four have had any seizures after the first injection. Unfortunately the twenty-eighth patient died. In addition to eclampsia she had a concealed accidental hæmorrhage and anuria. She seemed well twenty-four hours after delivery, but had a sudden attack of acute pulmonary œdema and died in a few minutes.

The incidence in epidemics of poliomyelitis, populations of all ages included, seldom exceeds one case per 1,000 of population, and among adults the incidence is usually much lower than this. In the three major epidemics which have occurred in New Zealand (1916, 1925 and 1937), the attack rate among adults aged twenty years and over was in each case in the neighbourhood of 0·1 case per 1,000 of population (Table I). The high incidence in the present series was probably due in part to the great opportunities for the transference of the infection, and in part to better facilities for the detection of mild and abortive infections, under conditions of army life.

SEASONAL INCIDENCE.

The epidemic lasted from November, 1940, to July, 1941, the maximum intensity being reached in April. These are the months in which the greatest number of cases occur in New Zealand also, in both epidemic and non-epidemic years; in the northern hemisphere, on the other hand, the incidence is commonly greater between May and December. This epidemic among New Zealanders on alien soil conformed in its seasonal distribution to previous experiences with poliomyelitis in New Zealand.

MORTALITY RATE.

Four deaths occurred among 40 subjects. This is a low mortality rate for poliomyelitis in adult New Zealanders. For example, in the epidemic of 1916, in one province for which reliable figures are available, there were 16 deaths among 57 infected adults of both sexes. In the epidemic of 1937, 59 cases occurred among adult males, and 15 of these were fatal. The low mortality rate in the present series may have been associated with the high morbidity rate and the high proportion of non-paralytic cases.

MULTIPLE CASES.

Fifteen cases occurred among 13 more or less widely separated units. The remaining 25 cases occurred in three battalions. On only two occasions did two cases occur in a single tent. In both instances the two men both became ill within a few days of each other; this indicates a probable common cause of infection rather than a direct subject-to-subject spread.

AGE.

The epidemic was remarkable for the fact that it arose in and remained confined to an entirely adult male population. We have been unable to find any record, in the limited literature to which we have had access, of any similar epidemic of poliomyelitis. In recent years, however, in different parts of the world, an increasingly large proportion of cases in epidemics of poliomyelitis has occurred among adults. For instance, in the 1934 epidemic in Denmark (3,938 cases), no fewer than 33·2% of all cases occurred among persons aged fifteen years and more. In the Californian epidemic of 1934, 31·8% of all cases occurred among persons aged fifteen years and over (Jensen, 1935).

In the last epidemic in New Zealand (1936-1937), the percentage distribution of the cases among the various age groups differed considerably from that recorded in the two previous epidemics (1916 and 1925). The morbidity rate per 1,000 of population among adults, however, remained substantially the same (Table I). In the present series the ages of all the patients were between twenty and forty years; 36 patients were aged between twenty and thirty years, and the remaining four patients were aged respectively thirty-three, thirty-two, forty and thirty-eight years. Variations in age within this twenty to forty years age group did not affect prognosis with regard to either death or the occurrence of paralysis. The ages of the four patients who died were twenty-two, twenty-four, twenty-five and thirty-eight years.

MODE OF TRANSMISSION.

Recent investigations (for example, Paul *et alii*, 1941) suggest that in some outbreaks of poliomyelitis house flies may carry the virus. In countries where bacillary dysentery is prevalent, its incidence affords a rough index of the fly population at any time. Moreover, if both dysentery and poliomyelitis are fly-borne diseases, it might be expected that the seasonal distribution of the two diseases would be similar. To see whether this was so in this instance, the incidence of poliomyelitis month by month during the epidemic was compared with the incidence of diarrhœal disorders occurring in the same months, and investigated at a New Zealand general hospital (Figure I). Clearly there was no correlation in this instance between the seasonal incidence of dysentery and that of poliomyelitis. The former showed the expected fall during the winter months from December to March, rising again with the onset of warmer weather in April.

The same lack of correlation between the two diseases was evident in individual battalions. In January, 1941, "X" battalion had six cases of poilomyelitis; but no patients suffering from diarrhœa were admitted to the hospital from that battalion in that month. In "Y" and "Z" battalions, in April, 1941, 16 cases of poliomyelitis and 29 cases of diarrhœal disorders occurred. Since these two units were composed largely of recent arrivals in the Middle East, this incidence of diarrhœa was not unduly high. However, four cases of poliomyelitis in which diarrhœa was a symptom occurred in these two battalions, and the explosive nature of this part of the epidemic suggests the possibility that the route of infection in "Y" and "Z" battalions may have been the alimentary tract.

SYMPTOMS AND SIGNS.

Table II sets out the symptoms and signs in the present series during the pre-paralytic stage.

Evidence of Neural and Meningeal Excitability.

Headache.—Headache was present in 39 cases. In one instance only was it absent; this patient was a man who was afebrile throughout and who had temporary weakness of the arms. It can be stated that in this series the patients who had milder degrees of headache more frequently had low numbers of cells in the cerebro-spinal fluid than those who had severe headache; but there is no conclusive evidence of a direct relationship between the severity of the headache and the degree of pleocytosis in the cerebro-spinal fluid.

TABLE I.[1]

Percentage Distribution of Cases, and Incidence per 1,000 of Population, of Poliomyelitis in Different Age Groups in New Zealand Epidemics, 1916, 1925 and 1937.

Age Group. (Years.)	1916.			1925.			1937.		
	Number of Cases.	Percentage.	Incidence per 1,000.	Number of Cases.	Percentage.	Incidence per 1,000.	Number of Cases.	Percentage.	Incidence per 1,000.
0 to 5	200	59·3	4·3	641	55·3	5·1	245	27·3	1·9
5 to 10	65	19·3	1·6	273	23·6	2·2	291	32·5	2·1
10 to 15	27	8·0	0·8	115	9·9	0·9	177	19·8	1·2
15 to 20	19	5·6	0·5	64	5·6	0·5	79	8·8	0·6
Over 20	26	7·7	0·12	66	5·7	0·08	104	11·6	0·10
Total	337	—	0·89	1,159	—	0·87	896	—	0·56

[1] The figures for 1916 refer to one province only, those for 1925 and 1937 to the whole Dominion.

TABLE II.

Group.	Signs and Symptoms.	Present. (Number of Cases.)	Absent. (Number of Cases.)	Not Recorded. (Number of Cases.)
Neural meningeal group.	Headache	39	1	—
	Backache	20	20	—
	Spinal rigidity	16	22	2
	Neck rigidity	25	14	1
	Kernig's sign elicited	16	21	3
	Pain in limbs	14	26	
	Muscle tenderness	7	31	
	Photophobia	17	23	
	Pain on eye movement	16	24	
Toxæmic group.	Fever	35	4	1
	Delirium	2	37	1
Gastro-intestinal group.	Dry tongue	24	8	8
	Vomiting	23	17	—
	Constipation	3	19	18
	Diarrhœa	6	34	—
Respiratory group.	Sore throat	8	32	—
	Cough	6	34	—
	Bronchitis	1	39	—
	Cervical gland enlargement	5	34	1

Tenderness of Muscles.—Tenderness of muscles was not a feature in this series; it occurred in only 17·5% of cases.

Comment.—In the present series, in cases in which moderately severe signs of neural and meningeal excitability were a feature, severe paralysis more frequently followed than in cases in which the signs were less severe. However, in no case could the outcome with regard to death or paralysis be foreseen by study of the early symptoms and signs. Thus, of 13 patients with severe symptoms, four escaped paralysis altogether, and of those with moderately severe symptoms, nine escaped paralysis.

Evidence of Toxæmia.

Fever.

Records of temperature are lacking in one case, and in four others at no time during the stay in hospital was the temperature recorded as being as high as 99° F. (The arbitrary figure of 99° F. is adopted as a definite indication of fever, in order to exclude possible variations due to the intake of hot fluids or solids, extremes of climate *et cetera*.) The average maximum temperature in 35 cases was 101·5° F.

Mode of Onset and Defervescence.—Twenty-two of the patients were admitted to hospital on the first, second or third day of the illness, and in all of these cases the onset was of progressive type. The remaining patients had all had symptoms for too long for an opinion to be formed. As the temperatures in general were not high, it was not possible to distinguish definitely between defervescence by crisis and defervescence by lysis. In almost all cases the termination appeared to be by lysis.

Height of Fever and Relation to Extent of Paralysis.—The height of the fever in 39 cases is shown in Table III, and from the same table it is evident that the maximum temperature was higher in the cases in which severe paralysis or death occurred than in the milder cases.

TABLE III.

Maximum Temperature Range. (Degrees Fahrenheit.)	Severe and Fatal Cases.	Slight Paralysis.	No Paralysis.	Totals.	Fatal Cases.
Under 99	0	1	3	4	0
99 to 100	1	1	7	9	0
100 to 101	1	1	5	7	0
101 to 102	3	0	2	5	0
102 to 103	7	0	3	10	3
103 to 104	3	0	0	3	1
104 to 105	0	0	1	1	0
Total	15	3	21	39	4

Duration of Fever.—Estimation of the duration of fever is difficult, as most of the patients had been ill for a number of days prior to their admission to hospital; this period varied between one and fifteen days, the average being four days. In the 35 cases in which fever occurred while the patients were under our care, the average duration of fever after admission to hospital was 3·2 days, the range being one to fourteen days. The relationship between duration of fever after admission to hospital, degree of paralysis and maximum elevation of temperature is seen in Table IV. It is evident that the duration of fever after admission to hospital tended to be longer in cases in which severe paralysis or death occurred than in those in which paralysis was transient, mild or non-existent. None of the patients who were severely paralysed or died were afebrile after admission to hospital; but one of 13 patients with mild paralysis and three of 21 patients without paralysis were afebrile. The mean maximum temperature was higher in the cases in which severe paralysis or death occurred.

TABLE IV.

Severity of Disease.	Number of Cases.	Duration of Fever. (Days.)	Maximum Temperature. (Degrees Fahrenheit.)
Severe paralysis, including four fatal cases	15	4·9	102·3
Slight paralysis	3	1·3	99·8
Non-paralytic	21	1·7	100·6
Fatal cases	4	5·8	102·9

Delirium.

Toxic delirium was noted in two cases. Both patients were admitted to hospital on the day of onset and the delirium was transient. The delirium had no apparent relationship to the meningeal irritation and the subsequent paralysis.

Evidence of Respiratory Lesions.

Symptoms related to the respiratory tract occurred in 14 cases of the series. Sore throat was the most common single symptom. Bronchitis occurred in one case only.

In our series there is no evidence that in cases in which upper respiratory symptoms and signs occur lesions of the bulbar nuclei are more likely to develop than in cases in which these symptoms and signs are absent. Of the 14 cases, bulbar lesions occurred in only two.

Enlargement of the lymph glands of the neck occurred in five cases. In no case did enlargement of axillary or inguinal glands occur. In one case the spleen was enlarged.

Evidence of Gastro-Intestinal Lesions.

Vomiting was present in 23 cases. It seldom occurred more than once or twice, and in no case was it an alarming symptom.

Diarrhœa occurred at the onset or soon after in six cases, and in five of these it was slight. From the history of the other patient it seems likely that he had bacillary dysentery. Four patients had both diarrhœa and vomiting. It is noticeable that none of the six patients who had diarrhœa developed paralysis.

Pain on movement of the eyeballs was noticed in 16 cases. This symptom seemed to bear no relationship to the maximum height of the temperature or to the severity of headache.

Paralysis.

Duration of the Pre-Paralytic Stage.

In the present series there were 19 patients with paralysis. The average duration of the pre-paralytic stage in these cases was 4·6 days, the minimum being one day and the maximum ten days. There was no relationship between the duration of the pre-paralytic stage and the degree of subsequent paralysis.

Stage of Paralysis.

Paralysis occurred in 19 cases. In seven the mode of onset of the paralysis was by widespread muscular weakness or by diffuse weakness of a whole limb, which soon became localized to certain muscle groups. In no case did diffuse weakness clear up completely without residual focal paresis. In the remaining cases the weakness was focal from the onset. The site of the paralysis is set out in Table V, in which a comparison is made with the large series quoted by Kinnier Wilson (1940).

TABLE V.

Site of Paralysis: Comparison with a Series of 5,748 Cases Quoted by Kinnier Wilson (1940).

Site of Paralysis.	Wilson.		Present Series.
	Number.	Percentage.	
Leg	4,519	78·6	13 out of 19
Arm	2,372	41·3	9 out of 19
Trunk	1,601	27·8	10 out of 19
Cranial nerves	767	13·3	5 out of 19
Throat, neck	333	5·8	Nil

Retention of Urine.

Retention of urine occurred in three patients. All three had paralysis of the abdominal wall, together with clinical evidence of severe involvement of the lumbo-sacral part of the cord. In no case were the pyramidal tracts involved, and in no case was any sensory loss observed. Reflex evacuation of the bladder did not develop in any of these cases. The duration of retention of urine in the three cases was fifteen, four and eighteen days respectively.

Clinical Types of Lesion in Cases of Paralysis.

Spinal.—In 12 cases the paralysis was of the ordinary spinal type; in two cases it was of the spinal "jump" type, with spread of paralysis from one limb to another after an appreciable interval.

Brain Stem.—In five cases there was involvement of cranial nerve nuclei; in two cases the spinal cord was involved as well. In the remaining three cases the brain stem alone was affected clinically. The palate was affected in four cases and the tongue in one case.

Abortive.—Twenty-one patients had typical symptoms and signs of meningeal reaction confirmed by the results of examination of the cerebro-spinal fluid. No cerebral, cerebellar or recurrent types of paralysis occurred.

TABLE VI.

Types of Disease and Mortality according to Types.

Type.	Incidence.	Deaths. N
Ordinary spinal	12	1
Spinal ascending	Nil	Nil
Spinal "jump"	2	1
Clinical evidence of cerebral involvement without spinal cord lesion	2	Nil
Cerebral and spinal involvement	3	2
Abortive, without serum	20	Nil
Abortive, with serum	1	Nil
Totals	40	4

Loss of Reflexes without Paralysis.

Of the 21 non-paralytic cases in the series, loss of reflexes (local or generalized) was observed in nine cases.

Duration of Paralysis.

In many cases paralysis was present on the patient's admission to hospital, and its duration before that is unknown. If patients with transient diffuse weakness and those who died are excluded, the shortest time within which focal weakness became present was five days. In many instances patients passed out of our observation before the paralysis had cleared up. In view of the belief that brain-stem lesions are transitory, it is of interest to note their duration in this series of five cases: (i) over six months, (ii) death ten hours after admission to hospital, (iii) death on the fourth day, (iv) more than twenty-three days, (v) more than thirty-five days.

Total Incidence of Paralysis.

In Table VII a comparison is drawn between the case incidence and the incidence of paralysis in a previous epidemic in New Zealand and in the present series. As has been pointed out above, the high case incidence in the present series is probably due to greater opportunity for infection in the army and to the fact that facilities for medical examination and rapid admission to hospital of suspected patients lead to the recognition of mild and abortive infections.

TABLE VII.

Series.	Number of Patients.	Number Paralysed.	Percentage Paralysed.
All ages; New Zealand epidemic, 1936–1937	845	610	72·2
Over 20 years; New Zealand epidemic, 1936–1937	93	68	73·1
Present series	40	19	(19 out of 40)

CLINICAL PATHOLOGY: CEREBRO-SPINAL FLUID.

Cell Content.

Cerebro-spinal fluid findings in the early stages are available in 38 cases. The range of cells in these was 11 to 1,300 per cubic millimetre. Two spinal punctures were performed in each of 29 cases. In 36 cases cell counts were made for the first time in the first ten days of the illness. In Table VIII the 40 cases are divided into four groups of ten; it is seen that in the cases occurring early in the epidemic, the number of cells in the cerebro-spinal fluid was lower than in later cases, and the cells were almost entirely lymphocytes. We suggest that this is due in part to the fact that in these cases the counts were made on an average three days later in the course of the disease. The records of lumbar puncture are insufficient to allow of the formation of any estimate of the duration of pleocytosis in the disease; but of 20 cases in which spinal puncture was performed in the third week or later, in six only did the count exceed five per cubic millimetre; in five out of nine cases in which spinal puncture was performed in the second week, the count was less than 10 cells per cubic millimetre; of 10 cases in which spinal puncture was performed between the fifteenth and twenty-first days of the illness, in only three did the number of

TABLE VIII.

Results of Cell Counts in Cerebro-Spinal Fluid.

Case Number.	Number of Counts.	Average Day of Count.	Range in Days.	Average Total Number of Cells per Cubic Millimetre.	Range of Total Numbers of Cells per Cubic Millimetre.	Average Percentage of Polymorphonuclear Cells.	Average Percentage of Lymphocytes.
1 to 10	7	6·6	2 to 10	93·6	30 to 250	1·0	99·0
11 to 20	9	3·1	1 to 5	297·4	19 to 945	72·6	27·4
21 to 30	10	3·4	2 to 6	268·0	11 to 675	69·5	30·5
31 to 40	10	3·2	2 to 5	300·2	42 to 1,300	84·6	15·4

cells exceed five per cubic millimetre. In one case the number of cells was still 44 per cubic millimetre on the thirty-ninth day of the disease. In general, the cell changes in this series conformed to the wide variations possible in this disease. Later counts invariably revealed lower numbers of cells and higher percentages of mononuclear cells.

Cell Counts and their Relationship to Subsequent Paralysis.

Cell counts were made in thirty-six cases before the onset of paralysis or on the first or second day of paralysis. It is of doubtful value to make deductions from a small series of cases; but in this series cell counts were no guide to whether paralysis was likely to develop. The two highest numbers of cells were counted in non-paralytic cases. The results of cell counts made on the second day of paralysis were higher than those of counts made in the pre-paralytic stage or on the first day of paralysis—an anomaly due no doubt to the small size of the series. The series suggests that the cell count gives no indication of whether paralysis will occur. In the four fatal cases the average number of cells was 298 per cubic millimetre, and there was no relationship between the number of cells and the mortality rate.

Protein Content.

The increase in protein content of the cerebro-spinal fluid, which is commonly described as occurring simultaneously with the fall in the number of cells, was by no means constant in this series. Of 37 cases in which there is a record of early lumbar puncture, in all except six the protein content was normal or slightly raised; in the six exceptions the protein content ranged from 50 to 100 milligrammes *per centum*. The records of 30 later lumbar punctures show that the protein content was over 40 milligrammes *per centum*; in four cases only the record was "marked increase by Pandy test". In only seven cases out of 29 in which more than one lumbar puncture was performed was there a significant difference in the protein content of the cerebro-spinal fluid as between the two punctures; in four of these cases the content was higher at the initial puncture.

Sugar Content.

The sugar content of the cerebro-spinal fluid, estimated in 19 cases in the acute stage, ranged from 35 to 110 milligrammes *per centum*, the average being 87·4 milligrammes *per centum*.

Chloride Content.

The chloride content of the cerebro-spinal fluid, estimated in 22 cases in the acute stage, ranged from 670 to 780 milligrammes *per centum*, the average being 737·3 milligrammes *per centum*.

Summary of Cerebro-Spinal Fluid Findings.

The findings in the cerebro-spinal fluid may be summarized as follows. In the early stages the number of cells present ranged from 11 to 1,300 per cubic millimetre. During the first few days examination of the fluid generally revealed that the cells were mixed, polymorphonuclear leucocytes predominating. When the fluid was examined again within the first few weeks, invariably a pronounced fall in the total number of cells had occurred, and the differential count usually revealed a change to predominance of lymphocytes. The height of the number of cells in the cerebro-spinal fluid afforded no clue to subsequent prognosis with regard to either paralysis or death. Of 29 cases in which a second examination of the cerebro-spinal fluid was made, in only three was a significant increase in the protein content found at the second examination as compared with the first. In 19 cases the sugar and chloride contents were normal. These findings are typical of poliomyelitis.

Blood Groups.

The blood groups were determined in 23 cases. The distribution was as follows: group AB, 4·4%; group A, 39·1%; group B, 4·4%; group O, 52·1%. These proportions are substantially those in which the different groups occur in European populations.

Diagnosis.

Every one of the 40 patients in this series had either a lower motor neurone paralysis of poliomyelitic type or typical symptoms and cerebro-spinal fluid findings. In the cases in which the paralysis was well defined, there was no difficulty in diagnosis. In milder cases of poliomyelitis estimation of paralysis may be difficult, and determination of the precise muscle group affected may be impossible. Changes in reflexes may help; but few of the muscles which may be affected have specific reflexes obtainable clinically. In this series no patient was considered to be affected by paralysis unless this symptom was present beyond reasonable doubt. In more than half the cases in this series paralysis was absent; in these 21 cases diagnosis rested on a consideration of the symptoms and the cerebro-spinal fluid findings.

In the middle of April, 1941, after a lull of nearly two months, during which there were no admissions to hospial for poliomyelitis, a sudden outburst of the disease occurred in two battalions—"Y" and "Z" battalions—occupying adjacent lines in one camp. In all, 16 cases occurred in an explosive manner between April 18 and May 2; thereafter no cases occurred from these units. The notable features of this group compared with the earlier cases were the low incidence of paralysis and the low mortality rate. (Table IX.)

TABLE IX.

Source of Cases.	Number of Cases of Paralysis.	Number of Deaths.
Cases from "Y" and "Z" battalions (16)	3	1
Remainder (24)	16	3
Total	19	4

Of the three cases in which paralysis was present, in two the disease was mild. The third patient died, and post-mortem examination revealed changes typical of poliomyelitis. A comparison of the symptoms and signs in the two groups reveals that the differences in the clinical picture were slight and were mainly in the direction of lesser severity in the "Y" and "Z" battalion cases compared with the remainder. In the former group five patients suffered from diarrhœa and vomiting, and in view of the explosive nature of the outbreak in this group it is of interest to speculate whether the alimentary tract was the site of infection. In this group also fever was less severe and of shorter duration than in the remainder, and symptoms of neural and meningeal irritation were usually milder. There was no significant difference between the cerebro-spinal fluid changes in the two groups.

Prognosis.

Mortality.

Four deaths occurred among the forty patients. The ages of the four patients who died were respectively twenty-two, twenty-five, twenty-four and thirty-eight years. Of the 19 paralysed patients four died. The low mortality rate in the present epidemic among adults is striking.

Two patients died on the fourth day of illness from medullary paralysis. One patient died on the thirteenth day with clinical evidence of persistence and spread of infection in the central nervous system. The fourth patient was tided over the acute stage by a Drinker respirator, but died thirty-five days later from toxæmia and respiratory embarrassment, consequent upon respiratory paralysis, pneumonia and empyema.

In general, in the fatal cases the maximum temperature was higher and the fever more prolonged after the

patient's admission to hospital than in the non-paralytic cases. The early signs and symptoms of meningeal irritation were pronounced, sometimes severe.

Paralysis.

Consideration of the early symptoms and signs and the cerebro-spinal fluid findings in any case in the present series gave no prognostic help. It is true that high and prolonged fever and severe symptoms of neural and meningeal irritability were more frequently followed by paralysis than were low temperatures and mild symptoms; but exceptions were so frequent that these deductions could not be applied to individual cases. A similar conclusion was reached by Tebbutt and Helms (quoted by Kinnier Wilson, 1940) in their study of the 1931-1932 epidemic in New South Wales.

PATHOLOGY.

The following is a summary of the pathological changes in the four fatal cases.

Nervous System.

In all four cases, in the meninges vascular congestion with or without a cellular exudate was present. In one case the membranes over the spinal cord were thickened by a fibrinous exudate. In levels of the nervous system above the pons seldom was any abnormality found other than vascular congestion, and even this was commonly absent. In two cases the cerebral cortex was infiltrated with inflammatory cells. In no case did microscopic examination of sections at these levels reveal nerve-cell degeneration.

In each case, in the pons vascular changes were present, and in two cases a cellular exudate was found as well. In one case nerve-cell degeneration had occurred at this level, though this was not very prominent. In the *medulla oblongata* vascular changes were present in all four cases, and in three cases cellular exudates and nerve-cell degeneration were present as well. The only subject in whom cell degeneration was absent at this level was one who died on the forty-ninth day of illness from bronchopneumonia, empyema and paralysis of respiratory muscles.

In the spinal cord pathological changes were present in the cervical, dorsal and lumbar segments in all four cases. In each case widespread infiltration with inflammatory cells was observed with extensive degeneration of the nerve cells of the anterior horns, amounting in some cases to actual necrosis.

Other Organs.

In every case cloudy swelling of heart-muscle, liver and kidneys was present. These changes were most intense, as would be expected, in the subject who died from bronchopneumonia, empyema and intercostal paralysis. This subject had a soft and friable spleen. Two patients who died on the fourth day from medullary paralysis had pulmonary œdema. In none of the cases were lesions of the gastro-intestinal tract present, and in none was enlargement of mediastinal or mesenteric glands found.

SUMMARY AND CONCLUSIONS.

1. An epidemic of poliomyelitis is described among an adult male population of the New Zealand Expeditionary Force in Egypt.

2. The epidemic was remarkable for its high morbidity rate (2·2 cases per 1,000 of population), for the high proportion of non-paralytic cases (21 out of 40) and for the low mortality rate (4 out of 40).

3. Forty cases occurred during a period of nine months. The seasonal incidence was similar to that of previous epidemics in New Zealand.

4. The distribution of the cases by months bore no relation to the monthly incidence of diarrhœal disorders during the same period.

5. The symptomatology was that commonly observed in epidemics of poliomyelitis.

6. The pre-paralytic stage lasted for one to ten days (average 4·6 days).

7. In paralytic cases usually fever was higher and more prolonged and symptoms of neural and meningeal excitability were more pronounced than in non-paralytic cases.

8. Paralysis occurred in 19 cases. The lower limbs were the commonest site of paralysis. There was a high incidence of paralysis of trunk muscles (10 cases), and in three cases the bladder was affected. No patients showed clinical evidence of involvement of structures higher than the *medulla oblongata*. One patient required treatment in a Drinker respirator; he died on the forty-ninth day of illness.

9. Counts of the cells in the cerebro-spinal fluid generally revealed a mixed polymorphonuclear and lymphocytic pleocytosis, the former type of cell preponderating in the early stages, the latter type in the later stages as the total number of cells fell. In only three cases did an increase in the protein content of the fluid accompany subsidence of the elevated number of cells. When the sugar and chloride contents of the cerebro-spinal fluid were estimated, they were normal.

10. The pathological features in the nervous system in four fatal cases were characteristic of poliomyelitis. The changes in other organs were those of toxæmia or were referable to the mode of death.

11. Three patients died from medullary paralysis and one from bronchopneumonia, empyema and intercostal paralysis.

12. In attempts to estimate the probable outcome in individual cases, no help was afforded by consideration of the early symptoms, the duration of the pre-paralytic stage or the cerebro-spinal fluid changes.

13. The clinical and pathological features of this epidemic among male adults differed in no important features from those observed in epidemics of poliomyelitis affecting mixed populations of children and adults of both sexes.

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BIBLIOGRAPHY.

Annual Report of the Department of Health, New Zealand, 1938.

C. Jensen: "The 1934 Epidemic of Poliomyelitis in Denmark; Preliminary Report on Epidemiology, Clinical Features and Convalescent Serum Therapy", *Proceedings of the Royal Society of Medicine*, Volume XXVIII, 1935, page 1007.

J. R. Paul and others: "Detection of Poliomyelitis Virus in Flies", *Science*, Volume XCIV, 1941, page 395.

S. A. Kinnier Wilson: "Neurology", 1940, Volume I, page 234.

A. H. Tebbutt and K. Helms: "A Report on the Epidemic of Poliomyelitis in New South Wales, 1931-1932", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1933, page 43; quoted by S. A. Kinnier Wilson, *loco citato*.

A CONTRIBUTION TO THE PROBLEM OF MASCULINIZATION.

By H. F. BETTINGER and HUBERT JACOBS,
Melbourne.

MASCULINIZATION of a normally developed woman is known to occur either (i) as part of a general disturbance of the endocrine equilibrium, often in the form of Cushing's syndrome, or (ii) as the result of a more or less well-defined neoplasm. In the latter group the most straightforward cases are those that are caused by an arrhenoblastoma of the ovary. It is recognized that these tumours arise from equivalents of male gonadal tissue and that they produce male sex hormones. The development of the masculinization syndrome and its disappearance after removal of the tumour are well understood.

Much greater difficulties are encountered in cases in which the disturbances are apparently produced by cells

of the type of the adrenal cortex. In a number of such cases a well-defined tumour of the adrenal cortex can be demonstrated, and in them the syndrome disappears promptly after removal of the tumour. However, this type blends without sharp demarcation into another, in which no tumour is found anywhere and in which the syndrome is usually ascribed to a hyperfunction or dysfunction of the adrenal cortex. With regard to the masculinizing effects of hormones produced by adrenal cortical cells, it is true that in such cases the urinary excretion of 17-ketosteroids is increased. But various authors have recently pointed out that by no means all the 17-ketosteroids are androgenic, and one has probably to assume in addition some disturbance of the endocrine metabolism. This links this part of the group up with the cases in the first group, and it is a fact that patients with minor degrees of masculinization show minor degrees of other endocrine disorders as well, although the cases usually do not come up to the specifications of Cushing's syndrome.

Adrenal cortical cells may, furthermore, give rise to neoplasms in the ovary. Since attention was drawn to the occurrence of adrenal rests in the ovary by Marchand many years ago, they have been observed often enough to suggest that they are the source of a certain group of ovarian neoplasms. However, there is the greatest difficulty in differentiating such tumours, on purely morphological grounds, from lutein-cell tumours. The latter neoplasms are thought to arise mainly from luteinization of granulosa-cell tumours, and fortunately in such cases enough non-luteinized granulosa can usually be found to establish the diagnosis. But if this is not the case a decision may be hardly possible. In the past, the attitude has often been adopted that, if the tumour produces male hormone, it is to be regarded as an adrenal tumour, and if it produces female hormones, as a luteoma. This is undoubtedly an easy but unreliable short cut. It should be borne in mind that the chemical relationship between testosterone and progesterone is very great indeed, and that in certain animal experiments injections of progesterone have had masculinizing effects.

With so many aspects of the problem of masculinization still under discussion, any observation that has some bearing on this subject seems worth recording. In addition, the case about to be presented was remarkable also from other points of view.

Report of a Case.

A girl, aged fourteen years, was first examined by one of us (H.J.) in February, 1939. She reported that a swelling of her abdomen had become noticeable about six weeks previously, and that the swelling had increased ever since. Neither pain nor vomiting had been associated with the swelling. There was no general feeling of weakness or sickness nor any disturbance of micturition. The menarche had occurred at twelve years; the menstrual periods had since been regular every twenty-eight days, lasting for six days, and they had not been painful. During the last four weeks the patient had had three fairly copious but not painful vaginal hæmorrhages. Apart from some breathlessness on exertion, she had no other complaint.

The clinical examination revealed a pale-looking girl with features of masculinization. The eyebrows and eyelashes were long and copious, and there was a distinct growth of hair on the upper lip, as well as on both sides of the face and around the chin. The pubic hair was copious, but not distinctly male in distribution. The breasts were poorly developed. The thyreoid cartilage was prominent and the voice deep. Examination of the thoracic organs revealed no abnormalities. The abdomen was filled by a large mass, that seemed semisolid in consistency. The external genital organs were of normal configuration, except for the clitoris, which was hypertrophic, being 1·5 centimetres in length. An X-ray examination of the pituitary region showed it to be within normal limits.

At operation on February 22, 1939, large amounts of yellowish, somewhat gluey fluid escaped from the initial incision. After their removal, a large multilocular cystic tumour of variegated colour was exposed. It occupied the position of the left ovary, was attached to the Fallopian tube, but to no other viscus, and was partly degenerated or necrotic. The tumour was removed together with the left Fallopian tube. The uterus and the other ovary appeared normal. The peritoneal cavity was then closed without drainage after aspiration of as much of the fluid as possible. The patient made an uneventful recovery and was discharged from hospital three and a half weeks after operation.

She was then not examined for more than five years, on account of the surgeon's duties with the armed forces. When reexamined on July 25, 1944, she reported that she had been completely well since the operation, she had had no illnesses of any kind, and had acted as a Red Cross blood donor on three different occasions. There were no traces of the masculinizing features observed five years earlier. The patient was of average height and weight, extremely good-looking, of a decidedly feminine type. The growth and distribution of hair on the face and in the axillæ and pubic region was now normal, the voice had returned to a female pitch, and the thyreoid cartilage was no longer prominent. Only the breasts were still little developed. An examination of the external and internal genital organs revealed no abnormality; the previously hypertrophic clitoris was now of normal size. There was no evidence of a recurrence of any symptoms or signs in December, 1945.

The routine microscopic examination of the tumour removed at operation suggested a possibly malignant pseudomucinous tumour of the ovary, but no further studies were carried out at the time. When the case came up for review after the interval of five years, a further attempt was made at correlating the clinical and pathological findings. Sections only from six blocks of the tumour were available for this study. The pathological examination had obviously to answer two questions: (i) What was the exact nature of the tumour? Was it actually malignant? (ii) Could anything be found that would explain the masculinization? (The prompt disappearance of all masculine features after operation left no doubt that they had been caused by the tumour.)

The examination of the slides leaves no doubt that the tumour belongs to the group of the pseudomucinous neoplasms. But the question whether it is an ordinary cystadenoma or a carcinoma is less easily answered. In

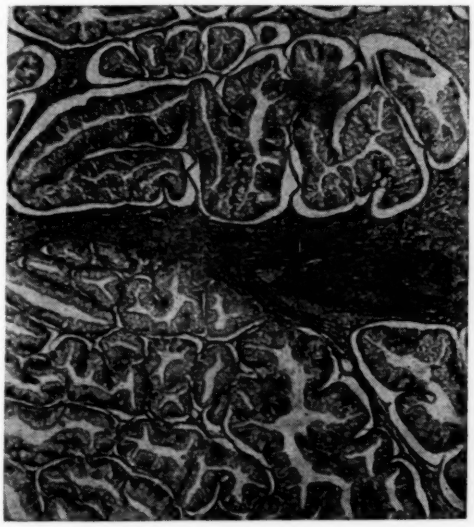

FIGURE I.
The glandular structure of the tumour at low magnification.

an ordinary cystadenoma one would expect much larger cystic cavities and much less epithelial proliferation. Most of the tumour is made up from glandular structures, often with extensive papillary epithelial formations. The epithelium itself is often of the typical picket-fence type, but equally often not so well differentiated, although retaining its polarity. Many parts of the tumour correspond to the *adenoma malignum* type of pseudomucinous cystadenocarcinoma that Barzilai has described in her "Atlas of Ovarian Tumours". Moreover, at least in a few places, such irregular microcystic formations are scattered

through the stroma, that the tumour has to be regarded as a carcinoma, although of low (grade I) malignancy (Figures I to V).

In regard to the second question, the only unusual finding is a peculiar variation in the structure of the

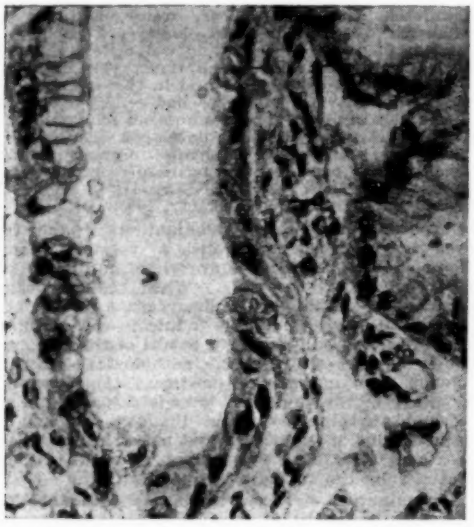

FIGURE II.
Regular and irregular epithelium in the same gland.

stroma of the tumour. Often the connective tissue is of the usual mature type. Long, fine or slightly coarse fibres with few long, thin nuclei are arranged in definite relationship to the epithelial structures. However, in other

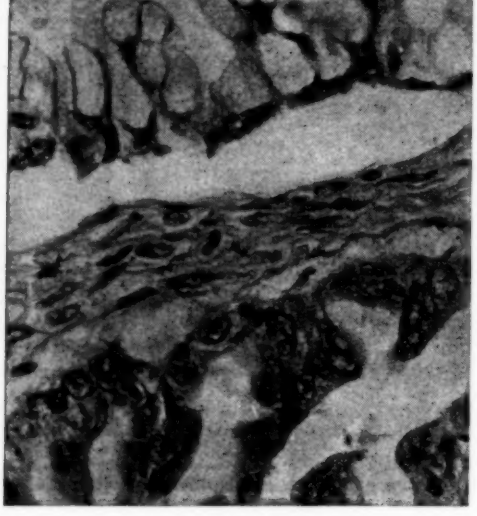

FIGURE III.
Regular, though somewhat proliferating, epithelium in one gland, irregular epithelium in the others.

places one finds substantial amounts of a cellular, hardly fibrillar tissue that is indistinguishable from luteinized *theca interna* (Figures VI to VIII).

Discussion.

Practically none of the standard textbooks on ovarian tumours contain references to similar observations. Only two exceptions have come to our knowledge. The appear-

FIGURE IV.
Beginning of dedifferentiation in one gland, higher degree of dedifferentiation in the other.

ance in Figure 305 in Ewing's "Neoplastic Diseases", fourth edition, is identical with numerous parts of our sections. The caption reads: "Adeno-carcinoma of the ovary with

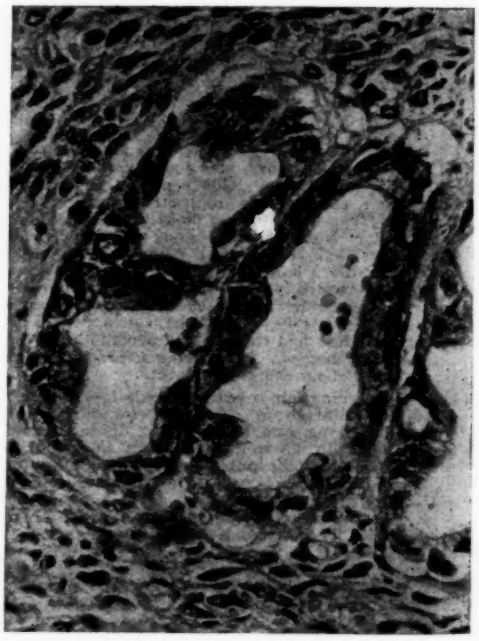

FIGURE V.
Irregular glandular structures in the stroma.

hyperplasia of interstitial cells" (italics are ours). Unfortunately, there is in the text, as so often in Ewing's book, no direct reference to the illustrations, the preceding page has only the following line: "The septa may contain

groups of hyperplastic interstitial cells of undetermined nature." Barzilai in her "Atlas of Ovarian Tumours" actually suggests that pseudomucinous tumours, on account of a luteinized stroma, may have endocrine effects, but she does not substantiate the suggestion by reference to actual observations. Somewhat more significant is a report by

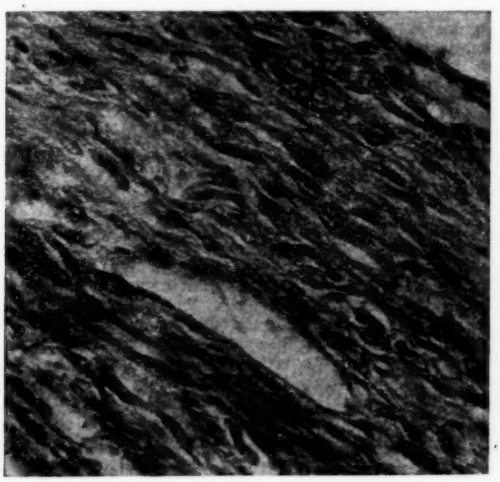

FIGURE VI.
The changes in the stroma: stroma of fibrous tissue type.

Geist and Gainer. They observed two women with pronounced masculinization, and in both there was no evidence of any of the usual causes of this condition, but both had enlarged ovaries which were subsequently removed. On microscopic examination an extensive luteinization of the *theca interna* was found. The authors discuss at some length the possible significance of this finding; they are

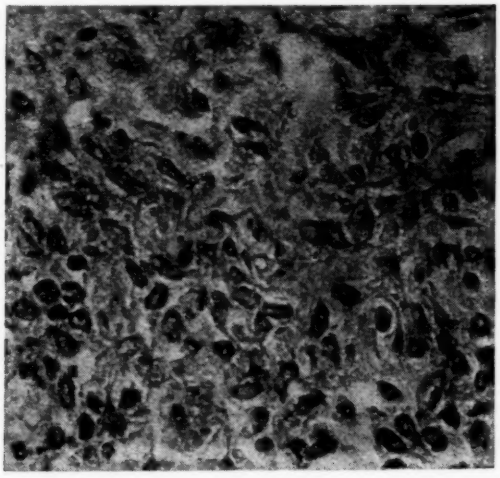

FIGURE VII.
The changes in the stroma: stroma of the type of the luteinized *theca interna*.

rather inclined to regard it as a feature secondary to some other non-disclosed disturbance, especially as no marked improvement resulted from the operations. Of great interest, however, is the observation of Turner. In his case of far-advanced masculinization a "microcystic degeneration" of the ovaries with luteinization of the *theca interna* and ovarian stroma was found. An operation resulted in the regression of all masculine and the restoration of full feminine features. Turner thinks that he obtained the favourable result because he did not remove the ovaries, but resected only the obviously abnormal tissue (about two-thirds of both ovaries) and so gave the remaining ovarian substance a chance to produce female hormones again and thereby induce refeminization.

The prompt disappearance of the masculinization after operation in our case leads us, in agreement with Turner, to believe that the changes in the ovarian stroma were responsible for the condition, at least as the last link of a chain. We have no certain evidence about earlier links in this chain, but we can safely rule out a general disturbance of the endocrine equilibrium as well as hyperplasia or tumours of adrenal cortical tissue, which are known to cause luteinization of the ovarian stroma. An action of gonadotrophic hormone producing luteinization of the ovaries and masculinization, as has been observed in experiments on rats, can also not be the cause in our case, because if this or any of the previously mentioned factors was responsible, they would still have been able to act through the remaining ovary.

It therefore remains only to assume that, on occasion, a pseudomucinous tumour of the ovary may initiate hormonal disturbances, as Barzilai has suspected.

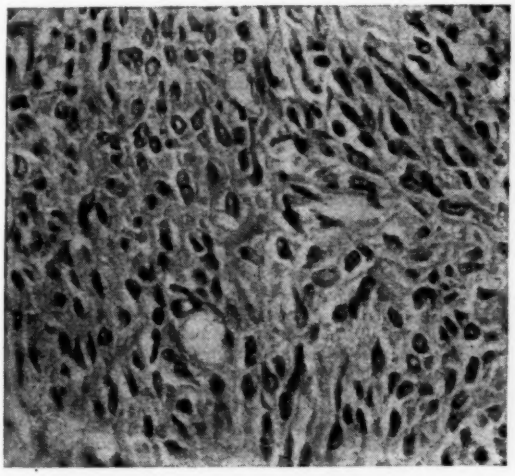

FIGURE VIII.
The changes in the stroma: an intermediate type.

Summary.

In summarizing, three points can be stressed:

1. The occurrence of a malignant pseudomucinous cystadenoma in a young girl and its six years' cure after simple removal.

2. The production of masculinization by hormones secreted by a luteinized *theca interna*.

3. The initiation of these hormonal disturbances by a neoplasm of the pseudomucinous group of ovarian tumours.

Bibliography.

J. Ewing: "Neoplastic Diseases", Fourth Edition.

G. Barzilai: "Atlas of Ovarian Tumours."

S. H. Geist and J. A. Gainer: "Diffuse Luteinization of Ovaries Associated with Masculinization Syndrome", *American Journal of Obstetrics and Gynecology*, Volume XLIII, 1942, page 995.

S. J. Turner: "Bilateral Microcystic Degeneration of Ovaries and Masculinizing Syndrome", *American Journal of Obstetrics and Gynecology*, Volume XLVI, 1943, page 298.

PURULENT MENINGITIS OF INFANCY AND CHILDHOOD: A TWELVE MONTHS' SURVEY OF THE RESULTS OF TREATMENT BY PENICILLIN.

By ELIZABETH K. TURNER, M.B., B.S.,
Medical Superintendent, Children's Hospital, Melbourne.

THE following report is presented with the object of demonstrating the efficacy of penicillin therapy in combating that formidable illness of childhood — purulent meningitis. The case records reviewed in this paper are those of children admitted to the Children's Hospital, Melbourne, during the period from March 1, 1944, to March 1, 1945, and summarize the first twelve months' experience of penicillin.

The types of meningeal infection discussed are those due to penicillin-sensitive microorganisms. These fall into two groups, as follows:

Group A: Gram-negative microorganisms: (i) *Neisseria intracellularis* (meningococcus).

Group B: Gram-positive microorganisms: (i) *Diplococcus pneumoniæ*, (ii) *Streptococcus hæmolyticus*, (iii) *Staphylococcus aureus*.

Not included in this paper is a large group of cases of purulent meningitis in which the causative organism was not determined. This failure may have been due to any of three factors: (i) late diagnosis, the organism having become more elusive and less amenable to culture *in vitro*; (ii) the empirical use of sulphonamide therapy before the child reached hospital, and the consequent rapid disappearance of the infecting microorganism from the cerebro-spinal fluid; (iii) inadequacy of laboratory test. In my opinion the second factor was of primary importance. Many of these children presented signs and symptoms typical of meningococcal meningitis, namely, pyrexia, purulent cerebro-spinal fluid, and a characteristic petechial rash. Such children, though included in a recent American survey,[1] are omitted here, all the diagnoses having been bacteriologically confirmed; but it is of interest to note that over 30 such patients were treated, of whom 22 patients required the intrathecal administration of penicillin to assure their recovery. Also, in this review the less tractable clinical type of meningeal infection due to the *Hæmophilus influenzæ* finds no place. Progress achieved at the Children's Hospital, Melbourne, in the specific therapy of *Hæmophilus influenzæ* meningitis is recorded in a communication to THE MEDICAL JOURNAL OF AUSTRALIA of March 3, 1945.

Age and Sex Distribution.

The graph (Figure I) depicts the age and sex distribution for all types of infection. There is a predominance of males over females, and greatest incidence occurs in the age group from birth to six months. This does not accurately represent the true state of affairs, as several children affected with purulent meningitis and belonging in age groups over three years were transferred to the Queen's Memorial Infectious Diseases Hospital, Fairfield, if their clinical condition permitted transport. These children are not included in this series.

Diagnosis.

It is a routine procedure in the casualty department that any child presenting with one or more of the following signs or symptoms, for which no adequate cause can be discovered, is subjected to a diagnostic lumbar puncture: (i) presenting symptoms, in order of frequency: vomiting, fever, drowsiness and lethargy, irritability, headache, convulsions, chills; (ii) presenting signs, in order of frequency: pallor, pyrexia, petechiæ, neck stiffness, general rigidity, collapse. As a result, many children have been admitted to hospital for treatment early in the course of their affection. The cerebro-spinal fluid is then examined by total and differential cell count. A direct smear is made and stained by Gram's method; in approximately 40% of cases the causative organism is detected. Cultures prepared from the cerebro-spinal fluid are incubated overnight, and confirmation of the type of organism is obtained in twenty-four hours.

Attempts at culture of microorganisms from the blood have been made in some cases, but not in all in this series. In several of the cases of fulminating meningococcæmia, in which death occurred in a few minutes to a few hours after the patient reached the ward, diagnosis was made by finding the meningococcus in a direct smear of the heart blood *post mortem*.

Management and Therapy.

Most children received one of the sulphonamide drugs immediately, by the mouth, or if they were unable to swallow, by the intravenous route. Choice of the sulphonamide was limited by the following considerations. Sulphapyridine and sulphathiazole were the only two available for parenteral use. Limited quantities of sulphamerazine were procurable. Sulphadiazine was available for oral use throughout, and was always given with alkali, because of experience of macroscopically evident hæmaturia in several patients treated with this drug.[2] The number of patients treated with each drug was as follows: sulphadiazine, 27; sulphamerazine, 18; sulphapyridine, 7; sulphathiazole, 4. Several of the patients received more than one type of sulphonamide preparation during the course of their disease. Thirteen patients received no sulphonamides.

The consensus of opinion is that sulphadiazine, sulphamerazine and sulphapyridine are equally effective therapeutically, but that sulphamerazine induces least toxic effects and is easiest of administration.

Criteria for the Use of Penicillin.

Clinically the cases would be divided roughly into two groups, A and B. Group A consisted of children whose history, with the clinical findings, suggested that the infection was subacute and that no appreciable progression of the disease would ensue during the three to eight hours required for the sulphonamide concentration in the blood to reach an effective therapeutic level. Patients in this group were treated with sulphonamides alone, and if the clinical response was not satisfactory after twenty-four to forty-eight hours, sulphonamides were either supplemented with or replaced by penicillin. A total of 16 patients recovered successfully on treatment with sulphonamides alone; all these had meningococcal infections.

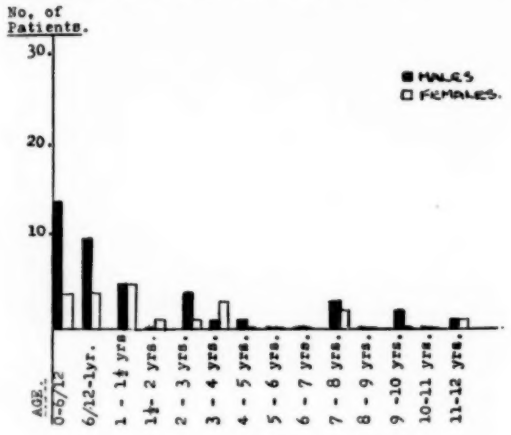

FIGURE I.

Group B consisted of children whose history, with the clinical findings, suggested an acute, fulminating or overwhelming meningococcal infection, in which the immediate institution of effective therapy was imperative, and of those affected with meningitis due to the pneumococcus, streptococcus or staphylococcus. This group comprised the chil-

dren suffering from fulminating meningococcæmia, from meningitis with septicæmia, and from acute meningitis with toxæmia and collapse.

Dosage and Routes of Administration of Penicillin.

Both the calcium and sodium salts of penicillin prepared by the Commonwealth Serum Laboratories were used. The working basis for dosage adopted was that of 1,000 units of penicillin per pound of body weight in the twenty-four hour period. Penicillin was administered by the parenteral route in the following dosages:

TABLE I.

Weight. (Pounds.)	Age (Approximate).	Dosage (Every Three Hours).
Under 8	Under 1 month.	1,000 units.
8 to 16	1 to 6 months.	2,000 units.
16 to 30	6 months to 3 years.	3,000 units.
30 to 40	3 to 6 years.	5,000 units.
	6 to 12 years.	10,000 units.
	Over 12 years.	15,000 to 20,000 units according to general development and physique.

Table I represents the minimal dosage requirements; many patients received much larger doses than those outlined above, with good effect.

In the treatment of septicæmia, penicillin was administered by the continuous intravenous drip method, after the calculated number of units had been dissolved in normal saline solution. As soon as the condition of the patient improved, the administration of the drug was continued by the intramuscular route, generally by intermittent injections every three hours of a solution containing 5,000 units of penicillin per millilitre, but occasionally by continuous intramuscular drip, the calculated number of units being dissolved in 100 millilitres of normal saline solution and administered over twenty-four hours. This last-mentioned method was productive of a more durable effect in older children.

As it has been shown that the concentration of penicillin in the cerebro-spinal fluid does not reach satisfactory therapeutic levels when penicillin is administered parenterally, except when enormous doses are given, the intrathecal route has been adopted.

Intrathecal Dosage.

Meningococcal Meningitis.

In meningococcal meningitis the intrathecal dosage on the patient's admission to hospital was as follows: age under three months, 10,000 units; age three months to three years, 15,000 units; age over three years, 20,000 units. The intrathecal dose was decreased by 5,000 units per day, and an average of four intrathecal injections on successive days was found to suffice.

Pneumococcal, Streptococcal and Staphylococcal Meningitis.

The initial intrathecal dose in pneumococcal, streptococcal and staphylococcal meningitis was the same as for meningococcal meningitis, but this dosage was maintained until the cerebro-spinal fluid was macroscopically clear. The dose was then reduced by 2,000 units per day until the cerebro-spinal fluid could be passed as clear upon microscopic examination.

The frequency of administration and the amount of the intrathecal dose are largely governed by the spinal fluid cell count, the result of attempted culture and the clinical response. It is necessary to continue intrathecal therapy for longer periods in meningitis due to the pneumococcus, streptococcus and staphylococcus than in the meningococcal infections.

Table II gives the average number of days during which intrathecal therapy was administered to children who recovered.

TABLE II.

Type of Infection.	Number of Patients Receiving Therapy.	Average Number of Days.	Frequency of Injection.
Meningococcal	10	4·9	Once a day.
Pneumococcal	8	13·75	Twice a day for the first three or four days, then once a day.
Streptococcal	1	19·0	Twice a day, including seven days both lumbar and intraventricular injection.
Staphylococcal	2	6·0	Twice a day for the first two or three days.

If response to therapy is slow, or if there is any indication that a spinal block is developing, two further apparently effective methods of treatment are available. The first is ventricular puncture, with the injection of 10,000 to 20,000 units of penicillin directly into the ventricle. One such puncture may suffice, but it has been necessary to repeat the procedure once a day perhaps for as long as seven days in a few ultimately successful cases. In an infant the procedure is not difficult, the ventricle being approached through the lateral angle of the fontanelle or through the fronto-temporo-parietal junction. The second

TABLE III.

Fulminating Meningococcæmia Without Meningitis.

Age of Patient.	Sex.	Duration of Symptoms.	Cerebro-spinal Fluid Findings.	Sulphonamide Used.	Administration of Penicillin.				Period in Hospital.	Result.
					Intrathecal.	Intravenous.	Intramuscular.	Total Dosage.		
3 months.. ..	M.	11 hours.	Fluid not examined.	Nil.	—	—	—	—	20 minutes.	Death. *Post mortem*, Waterhouse-Friderichsen syndrome.
3 years 1 month.	F.	12 hours.	33 polymorphonuclear cells per cubic millimetre.	Nil.	Twice.	Continuous drip.	—	100,000 units.	7 hours.	Death. *Post mortem* Waterhouse-Friderichsen syndrome.
1 year 6 months.	M.	2 days.	No cells.	Nil.	Nil.	Continuous drip.	—	30,000 units.	4 hours.	Death. *Post mortem*, Waterhouse-Friderichsen syndrome.
8 years	F.	18 hours.	No cells.	"M & B 693."	—	—	—	—	2·5 hours.	Death. *Post mortem*, Waterhouse-Friderichsen syndrome.
1 year 3 months.	F.	12 hours.	No cells.	"M & B 760."	—	—	—	—	7 hours.	Death. No post-mortem examination.
1 year 10 months.	M.	24 hours.	2 polymorphonuclear cells per cubic millimetre.	Sulphadiazine.	—	—	—	—	13 days.	Recovered. Culture from blood contaminated, but probably meningococci.
1 year 2 months.	F.	24 hours.	No cells.	Sulphadiazine.	—	6 days.	11 days.	595,000 units.	22 days.	Recovered.
8 months.	M.	12 hours.	No cells.	Sulphadiazine.	—	—	Once.	20,000 units.	20 hours.	Death. *Post mortem*, Waterhouse-Friderichsen syndrome.

method is the intrathecal or intraventricular administration of heparin. This method has only recently been employed, and whilst results are so far encouraging, conclusions drawn would be premature.

Varieties of Meningeal Infection.

Fulminating Meningococcæmic without Meningitis.

There were eight cases of fulminating meningococcæmia without meningitis. Two of the children recovered. Post-mortem examination was performed in all the fatal cases with the exception of one, and the typical adrenal apoplexy (Waterhouse-Friderichsen syndrome) was disclosed. In all cases cultures were obtained from the blood or the meningococcus was found in the heart blood *post mortem*. The cerebro-spinal fluid was not turbid, and the cell counts are recorded in Table III.

It will be seen from Tables III, IVA, IVB and IVC that penicillin was used in fulminating septicæmic cases and in acute and severe meningococcal infections, but that the mild, subacute or chronic infections responded satisfactorily to sulphonamides.

In all the cases of meningococcæmia without meningitis which terminated fatally, the typical Waterhouse-Friderichsen syndrome was present, and death occurred in spite of intensive intravenous penicillin therapy, supplemented with whole blood transfusions and adrenal cortical extract.

Pneumococcal Meningitis.

Penicillin, combined with sulphonamide therapy, appears to offer hope for the younger patients suffering from pneumococcal meningitis, the youngest child to recover in this series being aged no more than three months.

Streptococcus Hæmolyticus Meningitis.

From Table VI it is seen that the type of hæmolytic streptococcus was determined in three cases only. It proved in all three to belong to group A.

At first glance Table VI appears extremely depressing; but on examination it will be seen that of seven cases, four, if not five, were hopeless from the time of the patient's admission to hospital.

It was disappointing to find in the sixth of these cases a cerebro-spinal block and hydrocephalus developing, in view of the fact that the *Streptococcus hæmolyticus* had been eliminated from the cerebro-spinal fluid and the toxæmia overcome. In fact, this child was on the point of

TABLE IVA.

Meningococcal Meningitis with Meningococcæmia.[1]

Age of Patient.	Sex.	Duration of Symptoms. (Hours.)	Sulphonamide.	Administration of Penicillin.				Days in Hospital.
				Intrathecal.	Intravenous. (Hours.)	Intramuscular.	Total Dosage. Units.	
11 months ..	M.	10	Sulphamerazine.	3	24	9	790,000	26
9 years 2 months	F.	22	Sulphamerazine.	3	—	7	590,000	10
3 years	F.	36	Sulphamerazine.	—	—	—	—	14
6 months	M.	8	Sulphadiazine.	—	24	7	485,000	20
9 months	M.	12	Sulphamerazine.	3	48	5	385,000	13
8 years	M.	8	Sulphamerazine.	—	24	5	705,000	14
10 years 2 months ..	F.	24	Nil.	2	36	6	164,000	15
5 months	M.	12	Nil.	—	48	13	895,000	17
1 year 6 months ..	M.	24	Nil.	—	24	9	355,000	15

[1] In all the following cases the cerebro-spinal fluid was turbid and contained from 58 to thousands of polymorphonuclear cells per cubic millimetre; all patients recovered without sequelæ.

TABLE IVB.

Meningitis without Meningococcæmia: Acute and Severe.[1]

Age of Patient.	Sex.	Duration of Symptoms.	Sulphonamide.	Administration of Penicillin.				Days in Hospital.
				Intrathecal.	Intravenous. (Hours.)	Intramuscular.	Total Dosage. (Units.)	
11 months	F.	26 hours.	Sulphamerazine.	4	—	5	205,000	11
10 months	M.	24 hours.	Sulphamerazine.	3	24	4	242,000	14
3 years 5 months ..	F.	3 days.	Sulphadiazine.	6	—	10	1,167,000	13
1 year	F.	2 days.	Sulphadiazine.	11	3	12	1,936,000	27
2 years 9 months ..	M.	3 days.	Sulphadiazine.	10	—	12	1,902,000	15

[1] In all the following cases the cerebro-spinal fluid was turbid and contained from 58 to thousands of polymorphonuclear cells per cubic millimetre; all patients recovered without sequelæ.

TABLE IVC.

Meningitis without Meningococcæmia: Mild, Subacute or Chronic.[1]

Age of Patient.	Sex.	Duration of Symptoms.	Sulphonamide.	Days in Hospital.	Result.
1 year 2 months ..	M.	24 hours.	Sulphamerazine.	13	Recovered; no sequelæ.
4 years	F.	24 hours.	Sulphamerazine.	14	Recovered; no sequelæ.
5 years	M.	6 hours.	Sulphadiazine.	13	Recovered; no sequelæ.
2 years 2 months ..	M.	24 hours.	Sulphadiazine.	16	Recovered; no sequelæ.
1 year 4 months ..	F.	24 hours.	"M & B 693."	9	Recovered; no sequelæ.
6 months	F.	24 hours.	"M & B 693."	20	Recovered; no sequelæ.
1 year 2 months ..	M.	5 days.	Sulphadiazine.	24	Recovered; no sequelæ.
6 months	M.	2 days.	"M & B 693."	30	Recovered; no sequelæ.
4 months	M.	4 days.	Sulphadiazine.	40	Recovered; no sequelæ.
12 years	M.	2 weeks.	Sulphadiazine.	18	Recovered; internal strabismus.
8 months	M.	2 days.	Sulphadiazine.	12	Recovered; no sequelæ.
1 year 3 months ..	F.	8 days.	Sulphadiazine.	25	Recovered; developed post-basic signs and hydrocephalus.
6 weeks	M.	4 weeks.	Sulphadiazine and sulphathiazole.	29	Recovered; internal strabismus.
9 months	F.	4 days.	Sulphadiazine.	12	Recovered; no sequelæ.
7 months	F.	48 hours.	Sulphadiazine.	36	Recovered; no sequelæ.

[1] In all the following cases the cerebro-spinal fluid was turbid and contained from 58 to thousands of polymorphonuclear cells per cubic millimetre. No penicillin was administered.

being discharged home when she commenced vomiting. Heparin was not available, or the final result might have been otherwise.

I consider that patients suffering from primary acute hæmolytic streptococcal meningitis now have a reasonable chance of recovery.[1]

[1] Further experiences since writing this paper have been more encouraging. Two infants have successfully recovered from streptococcal meningitis, one child, aged one month, recovering from meningitis due to the *Streptococcus viridans*.

Staphylococcus Aureus Meningitis.

Table VII gives the results of treatment of three patients suffering from *Staphylococcus aureus* meningitis.

Comment.

It is interesting now to examine the records of patients suffering from meningitis who were admitted to the Children's Hospital, Melbourne, prior to March 1, 1944.

Since the introduction of sulphonamides in 1937 there have been 365 cases of meningococcal meningitis with 55

TABLE V.
Pneumococcal Meningitis.

Age of Patient.	Sex.	Duration of Symptoms.	Sulphonamide.	Administration of Penicillin; Number of Injections and Dosage.				Time Spent in Hospital.	Result.	Type of Infection.
				Intrathecal.	Intravenous.	Intramuscular	Total Dosage. (Units.)			
1 year	M.	2 days.	—	1	—	—	20,000	2 hours.	Death.	Primary acute.
3 months ..	M.	24 hours.	—	1	—	—	20,000	½ hour.	Death.	Primary acute.
7½ months ..	M.	2 days.	—	2	—	2	280,000	2 days.	Death; paralytic ileus.	Primary acute.
9 months ..	M.	2 days.	Sulphadiazine.	41	—	43	1,901,000	51 days.	Recovered; no sequelæ.	Primary acute.
7 months ..	M.	24 hours.	Sulphadiazine.	13	—	—	96,000	49 days.	Recovered; no sequelæ.	Primary acute.
8 years ..	F.	5 days.	Sulphamerazine.	3	—	8	480,000	13 days.	Recovered; no sequelæ.	Primary acute.
3 months ..	F.	5 days.	Sulphamerazine.	15	—	10	379,000	29 days.	Recovered; no sequelæ.	Primary acute.
9 years 2 months	F.	22 hours.	Sulphamerazine.	3	—	7	590,000	10 days.	Recovered; no sequelæ.	Primary acute.
1 year	M.	3 weeks.	"M & B 693."	1	—	—	10,000	24 hours.	Death; pyocephalus.	Primary chronic.
9 months ..	M.	2 weeks.	"M & B 760."	43	—	34	1,810,000	48 days.	Death; pyocephalus.	Primary chronic.
8 weeks ..	M.	2 weeks.	Sulphamerazine.	4	—	4	210,000	9 days.	Death; congenitally abnormal kidney.	Primary chronic.
4½ months ..	M.	1 month.	Sulphamerazine.	26	—	19	678,000	26 days.	Death; hydrocephalus.	Primary chronic.
9 months ..	M.	5 days.	Sulphamerazine.	14	—	17	836,000	34 days.	Recovered; no sequelæ.	Primary chronic.
3 years ..	M.	3 days.	Sulphadiazine.	10	5	12	1,000,000	36 days.	Recovered; no sequelæ.	Secondary to fractured skull.
8 years ..	M.	3 weeks.	Sulphadiazine.	11	—	13	784,500	21 days.	Recovered; no sequelæ.	Primary acute.

TABLE VI.
Streptococcus Hæmolyticus Meningitis.

Age of Patient.	Sex.	Type of Infection.	Duration of Symptoms.	Condition on Admission to Hospital.	Sulphonamide	Administration of Penicillin; Number of Inections and Dosage.				Duration of Stay in Hospital.	Result.
						Intrathecal.	Intramuscular	Intravenous.	Total Dosage. (Units.)		
3½ years ..	M.	Fulminating with septicæmia.	3 hours.	Moribund.	—	—	—	—	—	12 hours.	Death.
11 months ..	F.	Chronic.	3 weeks.	Decerebrate rigidity.	—	12	—	24	1,090,000	61 days.	Death; liquefaction of cerebrum.
4 months ..	F.	Secondary to infected meningocele.	10 days.	Poor.	Nil.	—	—	—	—	22 days.	Death.
17 days ..	M.	Secondary to umbilical sepsis and septicæmia.	13 days.	Moribund.	—	—	—	—	—	12 hours.	Death.
7 years 9 months	M.	Secondary to cerebral abscess.	Some weeks.	Moribund.	—	—	—	—	—	14 hours.	Death.
2 months ..	F.	Acute.	12 hours.	Good.	Sulphamerazine.	23	—	32	619,000	—	Cerebro-spinal fluid sterilized. Block and hydrocephalus developed.
3 months ..	M.	Secondary to mastoid infection.	2 months.	Generalized spasticity.	Sulphamerazine and sulphadiazine.	19	—	21	564,000	60 days.	Alive and well; intelligence doubtful.

TABLE VII.
Meningitis Due to the Staphylococcus Aureus.[1]

Age of Patient.	Sex.	Type of Infection.	Duration of Symptoms.	Condition on Admission to Hospital.	Sulphonamide	Administration of Penicillin; Number of Inections and Dosage.				Duration of Stay in Hospital.	Result.
						Intrathecal.	Intravenous.	Intramuscular	Total Dosage. (Units.)		
8 days ..	M.	Secondary to umbilical sepsis.	2 days.	Moribund.	"M & B 693."	—	—	—	—	4 hours.	Death.
1 year 8 months.	F.	Acute.	24 hours.	Fair.	All types without effect.	7	Every 48 hours.	9	950,000	26	Recovered; no sequelæ.
11 years ..	F.	Acute.	2 days.	Good.	Sulphadiazine.	5	—	8	916,000	13	Recovered; no sequelæ.

[1] The second patient in the foregoing table made a dramatic response to penicillin, after three types of sulphonamide had failed. Details of the cases are reported elsewhere.[3]

deaths—a mortality rate of 15%. From March 1, 1944, to March 1, 1945, there have been 29 cases of meningococcal meningitis (the ages of the children ranging from four months to twelve years) and no deaths. The death rate from meningococcal septicæmia has not appreciably altered since penicillin became available.

Eighty cases of pneumococcal meningitis are recorded prior to March 1, 1944, with 67 deaths—a mortality rate of approximately 84%. In the twelve months commencing March 1, 1944, there occurred 15 cases of pneumococcal meningitis with seven deaths.

The figures for streptococcal and staphylococcal meningitis are too small to be of any comparative statistical value, but the general impression is that the prognosis is much more favourable in these cases than formerly.

Conclusions.

1. Penicillin is indicated in the treatment of purulent meningitis due to penicillin-sensitive organisms, and particularly when the clinical condition and pathological findings suggest a severe or fulminating infection. Meningococcal, pneumococcal, streptococcal and staphylococcal meningeal infections demand the institution of penicillin therapy.
2. Penicillin therapy should in most cases be combined with adequate sulphonamide administration.
3. Penicillin therapy must be intensive and continued until the cerebro-spinal fluid findings and clinical condition indicate that the infection is overcome.
4. Penicillin must be administered directly to the affected part—that is, the meninges—and must therefore be introduced into the theca, through the lumbar or cisternal route, or into the cerebral ventricles.
5. Intensive and prolonged administration of penicillin causes no demonstrable toxic effects, nor have any untoward reactions occurred. A note may here be of interest, that not infrequently on the day following the first intrathecal injection of penicillin, an intensification of the turbidity of the cerebro-spinal fluid may occur. This appears to be due to cellular reaction to penicillin, but it is not attended by any harmful clinical effects and the cerebro-spinal fluid progressively clears with further administration of penicillin.
6. Intrathecal injection of heparin in any case of suspected spinal block, or when clinical response is disappointing, may prove to be a valuable supplement to sulphonamide and penicillin.

Summary.

A review of a series of cases of purulent meningitis due to penicillin-sensitive organisms is presented. Age and sex distribution, diagnosis, management and therapy are discussed. The criteria for the use of penicillin are outlined and tables of dosage and routes of administration are suggested. Tabulated lists of case records of meningitis due to the *Neisseria intracellularis* (meningococcus), *Diplococcus pneumoniæ* (pneumococcus), *Streptococcus hæmolyticus* and *Staphylococcus aureus* are presented and the results are discussed and compared with previous records of patients admitted to the Children's Hospital, Melbourne.

Some conclusions are drawn with regard to the efficacy and the essential benefit of penicillin therapy in such cases.

Acknowledgements.

My sincere thanks are due to Dr. Reginald Webster, pathologist to the Children's Hospital, and Major P. L. Bazeley, O.B.E., for much valuable help and advice, and to the sister and nursing staff of Ward 16 for their untiring interest.

References.

(1) D. Goldring and R. Maxwell: "Diagnosis and Management of Severe Infection in Infants and Children: Meningococcal Infections", *The Journal of Pediatrics*, Volume XXVI, 1945, page 1.

(2) E. K. Turner: "A Further Report on the Treatment at the Children's Hospital, Melbourne, of Influenzal Meningitis", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1945, page 219.

(3) E. K. Turner: "Penicillin in Pædiatrics: Preliminary Report of Cases of Varying Diseases Treated with Penicillin at the Children's Hospital, Melbourne", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1944, page 205.

Addendum.

Since March, 1945, the following patients have been admitted to the hospital and treated: (i) four patients suffering from meningococcal meningitis received penicillin, and all recovered; (ii) two patients suffering from pneumococcal meningitis received penicillin, and both recovered; (iii) two patients suffering from streptococcal meningitis received penicillin, and both recovered; (iv) one patient suffering from staphylococcal meningitis was still receiving treatment with penicillin when this paper was submitted.

Reports of Cases.

BILATERAL TRIGGER THUMB IN INFANTS.

By THOMAS F. ROSS,
Major, Australian Army Medical Corps.

IT is the purpose of this communication to report two cases of bilateral trigger thumb occurring in children, aged nine months and two years respectively.

Trigger or snapping thumb is a clinical entity due to *tendovaginitis stenosans* of the *flexor pollicis longus* tendon sheath at the base of the thumb. Its pathology is similar to that of trigger finger and De Quervain's disease[1][3] (*tendovaginitis stenosans* at the radial styloid process[1][4][11]), but it is the least common manifestation of this disease. Lipscomb[11] reviewed 190 cases of *tendovaginitis stenosans* and found only eleven cases of trigger thumb, all of which were unilateral. Involvement of both thumbs is still more uncommon. Compere[2] reviewed forty cases of trigger thumb and found only two in which bilateral involvement was present.

Whilst bilateral trigger thumb arising in adults is thus relatively infrequent, a review of the available literature indicates that it is rare in infants. Hauck[6] reported one case in a female infant. Compere[2] reported the case of a female patient, aged twenty years, whose symptoms commenced at the age of four years.

Case I.

A male child, aged nine months, was noticed by his mother to have constant flexion of both thumbs. Since birth the child could flex his thumbs, but could not actively extend them. Extension could be performed passively by his mother, who noticed that whilst the thumbs were extending there was an audible click, after which they suddenly snapped into full extension.

Examination disclosed similar findings in both thumbs. In each the terminal phalanx was held continuously in full flexion. At first considerable resistance to passive extension was present, but as greater force was applied, each thumb gradually extended until the resistance was felt to cease suddenly, so that the terminal phalanx jerked into full extension with an audible click. A small, non-tender swelling was palpable in front of the metacarpo-phalangeal joint of each thumb. It was situated in the line of the long flexor tendon and moved with it, giving an audible click on passive flexion and extension.

Operation was performed on each thumb through a small incision over the swelling. The tendon sheath was explored, and found to be constricted in front of the metacarpo-phalangeal joint by a thin, narrow, transverse band of fibrous tissue in the sheath itself. A longitudinal incision was made in the sheath, the constriction being divided and the underlying tendon, in which was a small fusiform swelling covered by pearly, normal-looking tendon, being exposed. The synovial layer was normal in appearance, and there was no fluid in the sheath. After division of the constriction, the tendon could be freely moved throughout its full range. No attempt was made to remove the swelling, and no tissue was removed from the sheath. The skin only was sutured.

The following day the infant was observed to have normal active movements of both thumbs. Similar findings were present eight months later, though a nodule was still palpable in each thumb. However, it had not increased in size.

Case II

This patient was a male child, aged two years. His mother stated that since the age of twelve months he had difficulty in extending his thumbs. He could actively bend both thumbs, though with some difficulty, but he could not straighten them without the passive assistance of his other hand.

Examination of each thumb disclosed a small swelling in the line of the long flexor tendon in front of the metacarpo-phalangeal joint. This swelling moved with the tendon, and an audible click was elicited on passive flexion and extension. When the terminal phalanx was fully extended, active flexion commenced smoothly and then suddenly stopped. Extra effort then appeared to jerk the tendon past an obstruction, after which flexion continued smoothly. From the fully flexed position, active extension started normally and then stopped completely, and no further extension could be obtained actively. When assistance was given by the examiner, there was felt at first a sense of firm resistance to extension. When this was overcome, an audible click was heard as the thumb was pulled into further extension. Extension was then able to be completed actively by the patient.

Incision over each tendon at the metacarpo-phalangeal joint disclosed the same macroscopic findings as in Case I. The constriction was divided in the same way, and the tendon was shown to have full movement.

After operation the patient was able to move his thumb normally. A follow-up examination six months later revealed normal, active movements of the thumbs. The small tendon nodules were still present, but had not changed in size.

Ætiology.

The ætiology of this disease occurring in infants is quite unknown. To say that it is congenital gives no clue as to its causation. A similar condition of trigger finger occurs in infants, and Watson-Jones[12] mentions a girl, aged four years, all of whose eight fingers were involved.

Repeated trauma is a constant factor in the disease in adults. This is especially seen in *tendovaginitis stenosans* affecting the *flexor digitorum profundus* tendons of the third and fourth fingers of untrained welders,[10] or the *abductor pollicis longus* and *extensor pollicis brevis* tendons at the radial styloid processes of young female factory workers who use rotary thumb movements in their occupation.[1][12] Obviously this factor can play no part in infancy. Even in adults Aitkin[1] points out that trauma is not the only cause, as in one of fifteen female factory workers suffering from De Quervain's disease, bilateral snapping thumb developed. However, it is significant that *tendovaginitis stenosans* at the radial styloid process has not been reported in infancy.

Pathology.

Little is written about the pathology of trigger thumb in children on account of its rarity, and the absence of any indication for removal of tissue. Macroscopic findings have been reported by Kroh[9] in the case of a child, aged three years, with involvement of one thumb only, by Hauck[6] in the case of two infants, one with unilateral and the other with bilateral trigger thumb, and by Compere[2] in his case, previously mentioned, in which bilateral trigger thumb had been present since the age of four years. From these findings and from those of the two cases reported here, the following conclusions may be drawn.

For some unknown reason, a constriction develops in the fibrous sheath of the *flexor pollicis longus* tendon at the base of the thumb. The synovial layer is not involved macroscopically, and there is no free fluid in the sheath. In the region of the constriction a fusiform swelling develops in the substance of the tendon. It is covered by normal-looking tendon fibres and synovial membrane.

This tendon swelling is probably a secondary reaction to the sheath involvement, as evidenced by the therapeutic success of mere division of the stenotic area. After this is divided, there is no further increase in size of the tendon nodule.

Even if this constriction is not divided, symptoms are only slowly progressive, provided the thumb is not subjected to trauma. This was evidenced in Compere's patient,[2] whose trigger thumbs were first noticed at the age of four years. The condition was only slowly progressive during the next sixteen years. Operation on each thumb then disclosed a nodule, one centimetre long, in the substance of the tendon in the region of the metacarpo-phalangeal joint. At this point the tendon, though normal in appearance, was swollen to twice its normal thickness. There was a dense transverse band of fibrous tissue in the sheath constricting the tendon at this region. The synovial membrane appeared normal. Compere excised the fibrous constriction of the sheath, and also a portion of the nodule after splitting the tendon over it. As a result of operation, full movement of the thumbs occurred. Seven months later there was no recurrence of symptoms. Microscopic examination of the excised nodule revealed numerous cartilage-like cells occurring in the dense fibrous tissue of the tendon.

The slow progress of this patient's lesion may be contrasted with the rapidly progressive picture of adult *tendovaginitis stenosans* when the thumb is subjected to the repeated trauma involved in certain industrial occupations.[1][10][11][12]

In the early stages, the pathology is similar to that of the condition in infants—namely, fibrous sheath constriction without visible inflammatory reaction[4] and with little change in the tendon itself.[12] With the continuation of trauma, the disease rapidly progresses, and one may find bulbous swelling (though not in every case[3]), or even grooving of the tendon.[1] The tendon may later become frayed or covered with granulation tissue.[4] The synovial sheath becomes reddened and loses its pearly lustre, and there may be adhesions between the visceral and parietal layers. Microscopic examination of the synovium may reveal hypertrophy and even fibrocartilaginous areas.[7] The fibrous sheath, at first a little thickened by fibrosis, may become three or four times thicker than normal in late cases, owing to dense fibrotic and cartilaginous changes.[4]

Even in these cases, treatment by simple incision of the constricting sheath leads to complete symptomatic relief, suggesting that the tendon involvement is secondary to the pathological changes in the sheath.

It may be seen, therefore, that *tendovaginitis stenosans* is essentially similar in infants and adults, but in the latter, any added traumatic factor will accelerate the rate and extent of the pathological process.

Symptomatology.

Trigger thumb may be noticed in infants at any time from birth onwards, according to the keenness of the mother's observation. Once present, symptoms are only slowly progressive, as has already been discussed.

Clinically, a small, movable, non-tender swelling is present at the base of each thumb in the *flexor pollicis longus* tendon. Each thumb can be completely flexed by active movement of the powerful long flexor muscle, though there is difficulty when the tumour is being pulled through the sheath constriction. When the swelling is proximal to the constriction, the less powerful thumb extensors cannot pull the swelling back through it without the aid of external passive assistance. Once the tumour is distal to the constriction, the rest of the range of extension can be completed actively. As the tumour goes through the constriction, the movement is jerky and an audible click or snap may be heard.

Treatment.

As in the case of adults, simple division of the constriction in the sheath is all that is required in the case of *tendovaginitis stenosans* in children. As the tumour is almost certainly secondary to the constriction, there is no indication for attempting its removal.[9]

This method of treatment gives complete symptomatic relief. These two patients were followed up for eight months only, but in this time there was no recurrence of symptoms and no increase in size of the tumours. On the other hand, the nodules did not decrease in size. This fact shows that once they develop, the pathological process is irreversible.

Prognosis.

It must be realized that, though incision of the sheath constriction relieves the pressure on the tendon so that the cause of tendon change is removed, nevertheless the actual pathological condition in the sheath is still present. However, Compere's case[2] shows that the progress of the lesion is so slow, even without operation, that it is not likely to cause further symptoms after incision. Should later occupational trauma throw stress on the affected tendons, it is possible that recurrence of symptoms will be seen. However, this is unlikely, as few occupations throw unwonted strain on the long thumb flexor tendons in contradistinction to the finger flexor tendons or the tendons at the radial styloid process. Even in the last-mentioned cases, once the sheath is incised, the same occupation can be followed without recurrence of symptoms.[1][10][11][12]

Summary.

Two cases of bilateral trigger thumb occurring in infants are presented.

Trigger thumb is due to *tendovaginitis stenosans*, but its exact ætiology in infancy is unknown, there being no predisposing recurrent traumatic factor as in the *tendovaginitis stenosans* of adults.

The lesion is localized first to the tendon sheath, and changes in the tendon itself are thought to be secondary to this.

The pathological process in children is similar to that in adults, but in the latter the added factor of trauma increases the rate and extent of the pathological process.

Symptoms and signs are shown to be due to the stenosis of the tendon sheath. They are completely relieved by simple incision of the constriction.

The prognosis as regards function is good, and the liability of recurrence of symptoms is practically nil.

Acknowledgement.

I wish to thank Major-General S. Roy Burston, Director-General of Medical Services, Australian Military Forces, for permission to publish this article.

References.

(1) A. P. Aitkin: "Stenosing Tendovaginitis at the Radial Styloid Process", *The New England Journal of Medicine*, Volume CCXXXII, January 25, 1945, page 105.
(2) F. L. Compere: "Bilateral Snapping Thumbs", *Annals of Surgery*, Volume XCVII, May, 1933, page 773.
(3) F. De Quervain: *"Ueber ein Form von chronischer Tendo vaginitis"*, *Correspondenz-Blatt für schweizer Aertzte*, Volume XXV, 1895, page 389.
(4) H. Finkelstein: "Stenosing Tendovaginitis at the Radial Styloid Process", *The Journal of Bone and Joint Surgery*, Volume XII, July, 1930, page 509.
(5) "Gray's Anatomy", Thirteenth Edition, 1893; quoted by D. C. Patterson and E. K. Jones, *loco citato*.
(6) G. Hauck: *"Ueber eine Tendovaginitis stenosans der Beugeschnensheide mit dem Phänomen des schnellenden Fingers"*, *Archiv für klinische Chirurgie*, Volume CXXIII, 1923, page 233; quoted by F. L. Compere, *loco citato*.
(7) W. Huber: *"Histologische Befunde bei der Tendovaginitis stenosans"*, *Virchows Archiv für pathologische Anatomie*, Volume CCXCI, 1933, page 745; quoted by P. R. Lipscomb, *loco citato*.
(8) F. Kroh: *"Schnellender Finger und Stenosierende Tendovaginitis der Finger Beugesehne"*, *Archiv für Klinische Chirurgie*, Volume CXXXVI, 1925, page 240; quoted by F. L. Compere, *loco citato*.
(9) T. P. McMurray: "A Practice of Orthopædic Surgery", Second Edition, page 317.
(10) N. Meadoff and A. B. Gray: "Stenosing Tendovaginitis", *Permanente Foundation Medical Bulletin*, Volume II, 1944, page 106; abstracted in *International Abstracts of Surgery*, December, 1944, page 477.
(11) P. R. Lipscomb: "Chronic Nonspecific Tenosynovitis and Peritendinitis", *Surgical Clinics of North America*, August, 1944, page 780.
(12) D. C. Patterson and E. K. Jones: "De Quervain's Disease", *The American Journal of Surgery*, Volume XLVII, Number 2, February, 1945, page 152.
(13) R. Watson-Jones: "Fractures and Joint Injuries", Third Edition, 1943, page 596.

Reviews.

ACUTE INJURIES OF THE HEAD.

THE second edition of Rowbotham's book on acute injuries of the head maintains the high standard of the previous edition.[1] It is a work in which the hard facts of diagnosis, treatment and pathology of head injuries are skilfully discussed and practically handled. The side of head injuries associated with a pathology which is not demonstrable is treated in a properly speculative and philosophical manner.

All sections of the book are excellently and engrossingly written and beautifully published. A surgeon seeking for practical guidance in a difficult subject will find it here. On the other hand, if he is seeking finality or enlightenment on the more abstruse and yet very present problems of post-head-injury syndromes, he may be disappointed. He will find a good presentation of our knowledge today—the lack is the fruit of our ignorance and means that there is much yet to be done.

This book should be very valuable to the general surgeon as well as to those who deal more exclusively in this branch of surgery.

[1] "Acute Injuries of the Head: Their Diagnosis, Treatment, Complications and Sequels", by G. F. Rowbotham, B.Sc. (Manchester), F.R.C.S. (England); Second Edition; 1945. Edinburgh: E. and S. Livingstone Limited. 9¾″ × 6¾″, pp. 440, with many illustrations, some in colour. Price: 30s. net.

AN INTRODUCTION TO INDUSTRIAL MEDICINE.

JAMES BURNET, a Doctor of Medicine and a Bachelor of Laws, has written a good introductory handbook for the study of certain aspects of industrial medicine and the legislation relevant thereto.[1] Sixty-three pages are devoted to industrial diseases and hygiene and twenty to workmen's compensation, truck and factory acts. In the first section the more common industrial poisons including metals and solvents are considered briefly but surprisingly well in view of the small size of the book. Then some "medical diseases" from anthrax to silicosis and compressed air illness are outlined, particular notice being given to "preventive measures". The final section on "Industrial Legislation" is well worth the attention of Australian readers. A useful provision is that in Part V of the *Factories Act* of 1937 relating to the notification and investigation of accidents and industrial diseases. This is a handy and inexpensive booklet which would be appreciated by practitioners who have patients engaged in factories.

THE PREMATURE BABY.

A SATISFACTORY book dealing with the premature baby has been written by Dr. V. Mary Crosse, who since 1931 has been senior medical officer in clinical charge of the premature baby ward at the City of Birmingham Maternity Homes.[2] From 1931 to 1943, 2,575 premature infants were treated in this ward. With this extensive experience we should expect a helpful book, and we are not disappointed.

After an introductory chapter on the definition and characteristics of a premature baby, the author proceeds to describe a general scheme of management before, during and immediately after labour. The main sections of the book then deal with institutional and home care, with clothing and methods of feeding, and with the complications that are liable to occur in the management of premature babies. Finally sections are given dealing with statistics.

The book gives the methods of care in great detail, illustrated by photographs, particular attention being devoted to the exact routine which must be followed by the staff if satisfactory results are to be obtained. All the recent advances in treatment have been incorporated, and references have been added at the end of most chapters. An index is supplied.

This book meets a long-felt need and will prove invaluable to obstetricians, pædiatricians, general practitioners and nurses.

MEDICINE FOR NURSES.

AFTER a number of reprints, Dr. W. Gordon Sears has produced a fourth edition of his book on medicine for nurses.[3] No major alterations have been made in the general form of the book, although it has been brought up to date with numerous small additions.

The book covers the syllabus laid down by the General Nursing Council in Great Britain, and aims to collect the essential facts of medicine for the nurse who is taking her course of medical lectures. We doubt very much whether any nurse will be able to absorb the pathological and theoretical sections in the book, as these demand a thorough knowledge of the fundamental sciences. However, the author in a book of this type is bound to keep one eye on the examination papers, and this type of knowledge is unfortunately being increasingly asked of the nurse. A chapter is included on *materia medica* and therapeutics.

The volume is clearly printed and is illustrated by numerous photographs and diagrams. An index is provided. The nurse will find the book a useful companion on the more theoretical side of medical nursing.

[1] "Outlines of Industrial Medicine, Legislation and Hygiene", by James Burnet, M.A., L.L.B. (London), M.D., F.R.C.P.E.; 1943. Bristol: John Wright and Sons Limited; London: Simpkin Marshall (1941) Limited. 7¼″ × 5¼″, pp. 92. Price: 7s. 6d.

[2] "The Premature Baby", by V. Mary Crosse, M.D. (London), D.P.H., M.M.S.A., D.R.C.O.G., with a foreword by Leonard G. Parsons, M.D., F.R.C.P., F.R.C.O.G.; 1945. London: J. and A. Churchill Limited. 8″ × 5¼″, pp. 164, with 14 illustrations. Price: 10s. 6d.

[3] "Medicine for Nurses", by W. Gordon Sears, M.D. (London), M.R.C.P. (London); Fourth Edition; 1945. London: Edward Arnold and Company. 7¼″ × 5″, pp. 462, with 67 illustrations. Price: 10s. net.

The Medical Journal of Australia

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FOOD CONSUMPTION IN AUSTRALIA.

In 1938 the Commonwealth Advisory Council on Nutrition, appointed by the Commonwealth Government, issued its final report. (See THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1938, page 614.) This Council sought to advise the Government on two aspects of the subject of nutrition: (*a*) the present state of nutrition of the Australian people; (*b*) the nature of any evidence that the Australian people were in any degree undernourished, or that their diet was improperly balanced or improperly prepared. In the concluding section of its report the Council stated that it might reasonably be assumed from the evidence reviewed that the Australian people were on the whole well fed, but that a minority was not obtaining and might not be in a position to obtain enough food. The Council could not determine the size of this minority, but in the limits of its survey the minority was represented by 6% of the dietaries recorded by housewives. The Council found that there was much ignorance in the community regarding the proper balance of food items, and that some people in both town and country were unable for various reasons to obtain the essential fresh foods. A considerable number of minor departures from normal health, generally described as malnutrition, were found among young children in both town and country. Medium and severe types of malnutrition were not found. Though the people of Australia were in 1938 regarded by the Advisory Council on Nutrition as "on the whole well fed", the state of affairs revealed by the report could not be regarded as satisfactory, particularly in view of Australia's rich endowment in the matter of food. Since 1938 the war has come and gone—the fighting has ceased. Though the Commonwealth has not suffered food shortages to an extent equal to those of many other countries, many irregularities, deficiencies and difficulties of many kinds have occurred. With the object of discovering whether the diet of Australian people has been affected by war-time conditions an investigation has been carried out by the staff of the Institute of Anatomy, Canberra, under the direction of the Nutrition Committee of the National Health and Medical Research Council.

A survey which covered every State of the Commonwealth, with the exception of the Northern Territory, and dealt with food consumption, was undertaken in 1944. The households investigated numbered 2,730 and comprised 15,235 persons. In New South Wales 150 households were investigated in the metropolitan area and 430 in the country; in Victoria 142 households were investigated in the metropolitan area and 437 in the country; in Queensland the figures for metropolitan households and country households were 227 and 458; in South Australia the figures were 105 and 201; in Western Australia 80 and 159; in Tasmania 70 and 134. The question will at once be asked whether the results of such a survey may be taken as representative of those that might be expected from the whole community. Except for the fact that the survey was limited to households containing two or more children, the selection of households was made at random in the areas chosen. The authors of the report admit that only limited importance can be attached to average figures for food consumption, because they mask the lower intakes that occur in some regions and in individual houses. Even if these considerations are borne in mind and if it is remembered that the survey covered approximately only 0·2% of the population of the Commonwealth, some important conclusions may be drawn. It would be simple enough to quote the general conclusion, suggested by the average figures, that, with the main exception of calcium, Australians during 1944 obtained supplies of foodstuffs and nutrients sufficient to ensure a fully satisfactory diet. This unfortunately would mean that the most interesting figures, those at each end of the scale, would be disregarded; it is these which are instructive. The authors of the report, after their detailed discussion, point out that though there is no lack of sufficient food, the levels reached are not uniformly satisfactory and that much improvement could be achieved in the quality of food intake in some sections of the community. The basic pattern of diet was found to be the same everywhere, and it was also found that the expenditure on food agreed closely with the levels of the diet in individual households. The average weekly expenditure on food throughout Australia per adult male unit was 18s. 9d., the average amount spent by those households which fully met or exceeded the levels of the recommended allowances was 21s. 10d. The average expenditure per adult male unit in those households falling short by the greatest amounts was 15s. 3d. Generally, it was found that the dietary level fell with the rise in the number of children in the family. This is what might have been expected. More deplorable is the finding that nursing and expectant mothers were at a serious disadvantage—failure to meet their nutritional needs was widespread. Some reference must be made to the different types of foodstuff. Bread and cereals were found to account for 18% by weight of the total mean weekly consumption for all households. The most noticeable finding was a relatively high consumption of bread and cereals in Tasmania compared with other areas and with the general average. As a matter of fact Tasmania shows up rather badly in the whole investigation and a special inquiry into the needs and resources of the island appears to be indicated. The consumption of fats other than butter has increased because of the introduction of butter rationing. Dairy products and eggs provided about 31% of the total mean weekly expenditure. Milk is the most important item in this group. Considerable variations in consumption of milk were found.

The highest was in Victoria, with the metropolitan area slightly in advance of country districts. The lowest was in Tasmanian metropolitan and country districts and in Western Australian country districts. For all States, excepting Tasmania and South Australia, the consumption of milk in country districts was less than in the metropolitan groups. The consumption of milk has increased since 1938, and this is of importance in regard to calcium intake. The consumption of eggs is higher in the country than in the city. Meat and meat products constitute some 12% of the total food consumption on a weight basis. The total consumption of meat in all areas was found to be remarkably level, the range being between 4·3 and 4·9 pounds per adult male per week. Although the total consumption of rationed meat does not exceed the ration allowance, more meat is consumed than before the war. This is thought to be accounted for by the increased consumption of non-rationed meat, poultry, rabbits and so on. Fruit and vegetables make up about 33% by weight of the total food consumed. With the exception of Tasmania, consumption was higher in metropolitan areas than in country towns. Special reference must be made to calcium. Throughout the whole of Australia the substance most lacking in the diet was calcium. In some 68% of households the consumption of foods was such that intake of calcium fell below the recommended allowances. The frequency with which such households were found, varied from 37·5% of the households in Sydney where mothers were in employment, to 84% of the households in Tasmanian country towns. The low intake of calcium is thought to have its origin in the low milk consumption found throughout all areas. Two factors in regard to calcium metabolism are mentioned. The first is that one of the factors governing the percentage absorption of calcium is the supply of vitamin *D* within the body of the individual. In Australia with its abundant sunshine most persons have adequate supplies of vitamin *D* in their bodies and for this reason the position in regard to calcium may not be quite so serious as it appears to be. Moreover, it has recently been suggested by McCance *et alii* that the amount of meat in the diet may affect the percentage of calcium absorbed, higher absorption occurring in persons with a high meat consumption. Australians are large meat-eaters.

In any consideration of this subject on the lines on which this report is drafted a great deal will be seen to depend on the efficiency of the mother as a manager. Although the authors of this report state that the expenditure on food agreed closely with the level of the diet in individual households, they admit that the housewife may play a large part through her efficiency as a manager. They mention two families in which adequate diets of the type described as "A" were obtained with the expenditure of 9s. or 10s. a week per adult male unit; home supplies of milk, eggs and vegetables were available at the same time. Other families spent up to 40s. per week per adult male unit and were able to attain only diets classed as "B", "C" or "D" grade. Again it is impossible to tell whether each member of the family receives his or her share of the amount of available food. In one large town in Australia which is addicted to excessive dog racing, it has been stated that the best cuts of meat are bought for the family's racing greyhound while the children are scantily clad and underfed. Where more money comes in because the mother is employed, more money was found to be spent on food and apparently on the right kind of food. Five recommendations are made. The first suggests that a special investigation of the factors connected with the intake and absorption of the calcium in Australian dietaries should be undertaken. The second suggests that a combined nutritional and sociological survey of Tasmania should be made. The third has to do with an education campaign for the public on nutrition, with special reference *inter alia* to expectant mothers. The fourth mentions the requirements of the vulnerable groups of the community in the distribution of milk. The fifth suggests that fruit and vegetables should be included in the number of price changes used by the court in the quarterly variations of the basic wage. With these recommendations we are in agreement. Unfortunately there is one statement in regard to the education of pregnant women to which we feel compelled to take exception. We agree that "one outstanding feature of the present era is that women endeavour to consult their medical practitioners much earlier in their pregnancy than formerly". We object, however, to the following words: "Unfortunately most medical advisers limit their advice to certain obstetrical facts and ignore completely the question of diet." This is a gratuitous *ex parte* statement which in our opinion is grossly exaggerated. No one with any experience of present day obstetrics could be guilty of such an exaggeration. No special recommendation is made about the question of transport for the distribution of foodstuffs—lack of transport is one of the main reasons why persons in widely scattered areas do not receive necessary foods, milk, fruit, and vegetables being chiefly affected.

The important aspect of this report is the inequalities revealed by it. To a large extent the food that people eat will depend on the amount of money that they have to spend. There are exceptions to this statement, but generally speaking it is true. We know which are the "protective" foods and we know which are the "vulnerable" persons in the community. It is not likely that good food alone will confer robust health on every person, but a person who is properly fed will be in much better condition to withstand adverse conditions than one who has not bothered about the protective elements in his diet. The taking of an adequate diet has been aptly described as the laying of a foundation on which abounding health may be based, and this is how the question should be regarded. Again it must be remembered that the framing of reports will of itself achieve nothing; they have to be followed by some kind of action. While on the whole Australians probably have reason to be satisfied with the war-time state revealed by the investigations that have been discussed, many would like to see another investigation directed to the discovery of ways in which help may be given to Great Britain and other countries in the matter of food.

Current Comment.

THE PROGNOSIS IN SUBARACHNOID HÆMORRHAGE.

THERE can be few, if any, conditions in medicine more sudden and dramatic than subarachnoid hæmorrhage. The patient is generally in normal health and is smitten suddenly with a most intense headache. The pain seems to strike at the base of the skull, and vomiting, dizziness and coma follow quickly. A good deal of interest has been taken in this condition since C. P. Symonds first described

it in 1924.[1] At the same time, not many reports covering large numbers of cases have been published. The condition is of the gravest import, but it is not invariably fatal. The blood found in the subarachnoid space at post-mortem examination may arise from several sources. Symonds in his paper of 1924 named the following four sources:

1. An effusion originating within the subdural space (as commonly results from traumatic laceration of the veins leading from the cerebral cortex to the great sinuses) may rupture the visceral layer of the arachnoid membrane and gain entrance to the subarachnoid space in this wise.
2. A hæmorrhage into the superficial parts of the nervous substances may break through the *pia mater* into the subarachnoid space.
3. A deeply situated cerebral hæmorrhage may rupture into one of the ventricles, and thence the effusion may find its way into the subarachnoid space through the roof of the fourth ventricle.
4. The hæmorrhage may be derived from one of the vessels lying in the subarachnoid space itself.

Of hæmorrhages from these four sources the last is the only type that should be described as spontaneous or primary subarachnoid hæmorrhage; the other three are secondary. The prognosis will depend on the size and site of the hæmorrhage and on the presence of any other pathological condition. In a series of 24 cases of spontaneous subarachnoid hæmorrhage reported by W. R. Ohler and D. Hurwitz in 1932,[2] 16 patients had an associated or complicating disease and 10 of them died. Only one of those with no complicating disease died. A much larger series was reported in 1943 by C. G. Magee.[3] He dealt with 150 cases of spontaneous subarachnoid hæmorrhage. The mortality rate in this series was 56%, and Magee remarks on the poor prospects of a really good recovery. He points out that the prospects diminish with advancing years. Few of his patients were entirely free from symptoms after their recovery, but these symptoms were often not a barrier to employment. Autopsy was carried out in 58 of Magee's cases. In 43 a ruptured aneurysm was found, and in the remainder the hæmorrhage was confined to the subarachnoid space, but no aneurysm could be detected. Magee examined the various symptoms and combinations of symptoms for prognostic indications, but without success. Magee's figures are further discussed by G. A. Wolf, junior, H. Goodell and H. G. Wolff,[4] who also deal with an interesting series of their own. Their series comprises 46 patients who were treated at the New York Hospital. With Magee's 150 cases the total is 196, and Wolf and his co-workers draw some general conclusions. They state that 29% of patients who enter hospital with subarachnoid hæmorrhage die during the first episode of bleeding; 14% die during recurrent bleeding between the second and the fourth weeks after the initial hæmorrhage, and an additional 5% die by the end of the first year. The majority of the remaining patients who survive the first year are alive three or four years after the initial hæmorrhage. The figures further show that some die in from one to twenty-seven years of a recurrence of the subarachnoid hæmorrhage; others die of some other illness. Though a few may live for twenty-seven years or longer after the first attack without recurrences, the threat of recurrent hæmorrhages and death is always present.

In the present state of our knowledge the prognosis of subarachnoid hæmorrhage is probably indicated by the figures that have been quoted. Whether an improvement can be effected remains to be seen. Wolf and his collaborators discuss the work of Dandy on the surgery of intracranial aneurysm. They believe that with an improvement in neurosurgical technique a reduction in mortality rate can be expected. The temptation to quote some of Dandy's figures must be resisted and the subject of surgery in this condition would also call for reference to the pathology—a fascinating but lengthy aspect of the question. In the circumstances, if we bear in mind the situation of the lesions and the pathological changes involved, scepticism on the benefits that may follow surgery in the days to come is surely excusable.

[1] *The Quarterly Journal of Medicine*, Volume XVIII, 1924, page 93; *Guy's Hospital Reports*, Volume LXXIII, 1923, page 139.
[2] *The Journal of the American Medical Association*, Volume XCVIII, 1932, page 1856.
[3] *The Lancet*, Volume II, 1943, page 497.
[4] *The Journal of the American Medical Association*, November 10, 1945.

CONGENITAL DEFECTS IN INFANTS AFTER MATERNAL RUBELLA: FURTHER REPORTS AND DISCUSSIONS.

THE literature on the occurrence of congenital defects in infants after the mother has suffered from rubella during the early days of her pregnancy is growing, and recent reports on the subject are of interest. J. C. Long and R. W. Danielson report the occurrence of defects in six infants following maternal rubella.[1] The mothers had contracted rubella when they were from two to six weeks pregnant. Three of them did not know that they were pregnant when the exanthem appeared. The disease was mild and was regarded as trivial. Three of the babies had bilateral cataracts which were associated with bilateral microphthalmos. In one instance the bilateral cataracts were membranous; the other cataracts, bilateral and unilateral, were nuclear and conformed to the original description given by Gregg. In three fundi which could be seen lesions were detected. All six children had cardiac defects. In addition the following lesions were noted: *talipes valgus*, cryptorchism, hypospadias, dacryostenosis. In discussions on this subject it has been suggested that clinicians should report cases in which a mother who has suffered from rubella during the early days of her pregnancy gives birth to a healthy baby. In this regard it may be noted that in one of Long and Danielson's cases the mother transmitted the rubella to a neighbour who was three and a half months pregnant. Her child was stillborn as the result of an obstetric accident. The baby's heart was found at autopsy to be normal, but unfortunately the eyes were not examined. Rubella at so late a stage of pregnancy as three and a half months is not so likely to be followed by lenticular changes as by deaf mutism.

C. H. Albaugh, of Los Angeles, has reported nine cases of congenital abnormality in infants following an exanthem in the mother in the early weeks of pregnancy.[2] In eight of the nine cases bilateral cataract was present and in eight congenital heart lesions were present. The infant without cataract had a heart lesion and also strabismus, and the mother was affected by rubella in the tenth week of pregnancy. In only three cases was microphthalmia present. In one of the cases of bilateral congenital cataract the exanthem affecting the mother was morbilli, and this occurred in the eighth week of her pregnancy. In another of the cases the mother was "exposed to morbilli" during the fourth week of her pregnancy. Albaugh, it may be remarked, gives a first rate review of the literature. In this he differs from Christopher Bull, who, in discussing the effect on the fœtus of diseases of the mother during pregnancy, creates a wrong impression.[3] Evidently he has not seen Gregg's first paper presented to the Ophthalmological Society of Australia (British Medical Association) in 1941 and published in its *Transactions* in 1942. Bull's first reference to the Australian literature is to the work of C. Swan *et alii*, published in this journal in September, 1943. He states that "another Australian investigator (Gregg)" supported Swan's work with a report of 78 cases of congenital cataract. Gregg, of course, was first in the field; it was the series of 78 cases which formed the basis of his first paper. Cataracts are not the only abnormality that may appear in the infant; others include cardiac defects, deaf-mutism and microcephaly. When available data are held to suggest that 100% of mothers who contract rubella in the first two months of pregnancy and 50% of those who contract it in the third month will give birth to infants with congenital anomalies, the importance of the subject is obvious.

[1] *Archives of Ophthalmology*, July, 1945.
[2] *The Journal of the American Medical Association*, November 10, 1945.
[3] *Archives of Pediatrics*, July, 1945.

Abstracts from Medical Literature.

DERMATOLOGY.

The Treatment of Sycosis Barbæ by Penicillin Cream.

A. BURROWS, B. RUSSELL AND H. B. MAY (*The British Journal of Dermatology and Syphilis*, May-June, 1945) define *sycosis barbæ* as a chronic staphylococcal folliculitis of the beard areas. On ætiological and clinical grounds, *sycosis barbæ* may be divided into two main types: (i) straightforward folliculitis; (ii) the seborrhœic type, an obvious extension of seborrhœic dermatitis. Sycosis has long been known as one of the most intractable diseases of the skin; often it almost disappears, only to flare up again at a later date. The use of sulphonamides gave fair results in a few cases, but penicillin appears to have opened up a new chapter in the therapy of sycosis. The duration of the infection in the 21 cases studied by the authors varied from fourteen years to eight weeks. Penicillin cream was employed rather than the solution, because it was more convenient to apply and more lasting in its effects, and because it produced better results. In all cases bacteriological examination was made of the pus from an infected hair follicle or from beneath a crusted lesion. All treatment was stopped for forty-eight hours before the patient attended at the laboratory for the taking of pus for examination. The pus was plated on plasma agar and blood agar. By the use of the former medium, coagulase-positive staphylococci could be easily picked off after sixteen hours' incubation, even if coagulase-negative staphylococci were also present. For the primary cultures sensitivity tests were put up by the antiseptic cream method (May and Stern, 1944). A staphylococcus strain was recorded as sensitive if the zone of inhibition on the plate was of more than half the diameter obtained with the standard Oxford H staphylococcus. If the diameter of the zone of inhibition was less than half this diameter, the strain was recorded as partially sensitive. When the predominant organism was penicillin-sensitive, the patient was provided with a pot of penicillin cream containing 200 units of penicillin per gramme. The cream was prepared by a modification of the method of Clark *et alii* (1943). Aseptic removal of the cream from the jar is necessary. A knife or spoon can be sterilized by being boiled in water for three minutes. The cream is removed from the jar and spread onto clean lint and thence thinly applied to the affected area twice a day. It is essential to test the sensitivity of the causal organism to penicillin before treatment is started. All infections recorded as due to strains of staphylococcus sensitive to penicillin were much reduced by the application of penicillin cream containing 200 or 400 units per gramme, except in one case, in which the reduction was slight. Culture in this case produced a mixed growth of *Staphylococcus aureus* and *Bacillus proteus*, the latter organism having an inhibitory action on the penicillin. The authors give the patient detailed instructions on paper regarding the aseptic handling of the cream. To lessen the tendency to relapse, it appears necessary to apply penicillin cream to the nares as well as to the skin of the beard, to treat nasal discharges, blepharitis, *otitis media* or *otitis externa*, and any manifestations of the seborrhœic state; to eliminate foci of sepsis in the throat, teeth and sinuses; to continue treatment for a few weeks after apparent cure; to use the cream prophylactically after relapses, or if nasal swabs persistently contain the offending organism; to induce the patient to discard infected shaving brushes and face flannels, or sterilize them by boiling, or use a brushless shaving cream; and to consider in severe cases the use of X-ray epilation in conjunction with penicillin treatment.

Skin Eruptions due to the Application of Sulphonamides.

C. A. GRANT PETERKIN (*The British Journal of Dermatology and Syphilis*, January-February, 1945) states that a patient with a skin eruption due to sulphonamide therapy nearly always gave a history of having had the sulphonamide applied externally, usually in the form of sulphanilamide powder, but sometimes in the form of a 10% or 20% ointment. In most cases, after several days' treatment a burning sensation was noticed in the lesions being treated; this was followed in twelve to twenty-four hours by an eruption over the face and neck. In the milder cases this was merely an erythemato-papular rash of a peculiar maroon colour, but in the more severe type the whole face, the nose and ears included, but not the shadow under the chin, became œdematous and later completely covered with huge golden-yellow crusts, the lips and eyelids being swollen. The eruption extended to the exposed parts of the neck and chest. Within a few hours of the appearance of the facial eruption a rash appeared on the other exposed areas—namely, hands, arms and knees. This rash was erythemato-papular in mild cases, but vesico-bullous in severe cases, in which often a high temperature and general toxæmia were also present. Some patients had been given sulphonamides by mouth to counteract the acute infection; this exaggerated the condition and produced as well a generalized morbilliform rash, followed by erythrodermia. The author encountered 65 cases amongst the troops in North Africa of light eruption due to external application of sulphonamides. Of these 65 patients, 32 had been treated for impetigo, eight for impetiginized seborrhœic dermatitis, three for septic infection of the limbs, five for "running ears", two for secondarily infected tinea of the feet, and fifteen for wounds and burns of the trunk. In 61 of the 65 cases sulphanilamide powder was the first sulphonamide drug to be applied. The author suggests that the eruption is almost invariably preceded by application of the powder, and that the patient becomes sensitized to the drug by its inhalation. It is urged that powdered sulphanilamide should not be applied to the skin for minor conditions. In North Africa, over 200 patients, including 184 suffering from impetigo or impetiginized seborrhœic dermatitis, were treated with 5% sulphathiazole in Lassar's paste or "Lanette Wax" cream, and their skin was freely exposed to light. Only one patient with respirator dermatitis developed the eruption described; this was mild and soon subsided. The author considers that 5% sulphathiazole in a suitable base is probably as safe as such drugs as ammoniated mercury for dermatological therapy.

Dermatitis Exfoliativa following Arsphenamine Therapy.

M. J. COSTELLO AND S. LANDY (*The New England Journal of Medicine*, March 29, 1945) report fifty cases of *dermatitis exfoliativa* following arsphenamine therapy, which occurred between the years 1937 and 1942. The average stay in hospital was forty-three days, the shortest four days and the longest 171 days. The average duration of the dermatitis was sixty-nine days. Forty-eight patients had their condition improved or were cured, and two died. On admission to hospital, one patient had primary syphilis, 34 were in the secondary stage, three had early latent syphilis and eleven had late latent syphilis. Forty-two patients had received neoarsphenamine, two had received "Mapharsen", two had received "Mapharsen" followed by neoarsphenamine because they developed mild dermatitis from the former, and two received acetylglucoarsphenamine. The type of arsenical administered to the remaining two patients was unknown. The majority of patients complained of pruritus or dermatitis soon after the fifth, sixth or seventh injection. In many cases either the warning signs were not observed or the patient was not questioned about them. Ignorance of the significance of mild dermatitis or mild itching in the course of arsenical therapy led to full-blown *dermatitis exfoliativa*. A number of the patients had received injections of neoarsphenamine some years before. Severe complications of arsenical therapy can usually be anticipated, but several of the histories show that in patients sensitized by previous injections of an arsenical preparation, the eruption appeared after the first injection. Robinson and Moore have induced a second attack of dermatitis with a small dose of arsphenamine—0·1 millilitre of a 1 in 2,000 solution—injected intradermally six years after the first eruption. One patient developed a mild eruption after twelve injections of "Mapharsen"; then neoarsphenamine was substituted. After five injections of the latter the patient eventually developed a generalized eruption. Probably the "Mapharsen" sensitizes such patients to the neoarsphenamine subsequently administered. "Mapharsen" was the cause of an arsenical eruption in two cases. "Mapharsen" seldom causes arsenical dermatitis. All the patients who developed an eruption after the first or second dose of an arsenical preparation gave a history of having previously received an arsphenamine. Twenty-five patients had a temperature of more than 100° F. Clinical jaundice occurred in two cases. Examination of the urine revealed no abnormality in most cases. In the great majority of cases studied the eruption was generalized and universal. In order of frequency the regions of the body affected were as follows: extremities, torso, axillæ, groins, neck, face, chest, back, eyelids, hands, feet, ears, wrists, ankles, scalp, lips, oral mucous membrane and conjunctivæ. General adenopathy was present in most cases.

Pruritus, burning and pain in varying degrees were common to all the eruptions. Œdema, especially of the lower extremities, due to stasis, and angioneurotic œdema of the eyelids, face, lips and mucous membranes, were constant features of the early manifestations of dermatitis. Peeling of the palms and soles accompanied by pruritus and fever was an occasional forerunner of the severe generalized eruption. In many cases, if these signs had been heeded, the generalized eruption might have been prevented or its gravity lessened. The eruption began with flexual erythema and pruritus, followed by large-sized, dull red, slightly elevated lesions on the torso, especially the back, and often resembled *erythema multiforme*, and by coalescence became plaque-like, lichenified and scaly. Vesico-bullous lesions occurred in the explosive types, especially when the patients had been sensitized by previous medication. Lowered resistance led to bacterial infections such as furunculosis, purulent conjunctivitis and blepharitis. Sodium thiosulphate was administered intravenously. The highest number of injections was ten. The patients also received one gramme of sodium thiosulphate by mouth three times a day. This drug was of no value. Intramuscular injections of calcium gluconate were of questionable value. Glucose solution was administered intravenously to ten patients daily in amounts varying from 20 to 50 millilitres of a 50% solution, to 1,000 millilitres of a 5% solution, the administration being continued for ten days. Glucose administered in this way appeared of benefit. Concentrated vitamin preparations were administered. Patients suffering from exfoliative dermatitis should not be given intramuscular injections of liver, because of the likelihood of abscess formation. Local treatment consisted of potassium permanganate and starch baths, inunction of boric ointment and compresses of Burrow's solution (one in ten). Secondary infection was treated by wet boric acid compresses and wet dressings of a 1 in 1,000 solution of silver nitrate. Crude coal tar paste was of value on the dry, patchy areas. The patient should be asked at each visit whether he has itching, fever, a rash or headache. The whole of the skin should be examined before the sixth, seventh and eighth injections when arsphenamine, neoarsphenamine or silver arsphenamine has been used. A similar examination should be made before the twelfth and fifteenth injections of "Mapharsen". If a rash of any sort develops and is not readily recognizable, it is safer to withhold treatment. The patient should be asked whether he has had previous injections. Further treatment with an arsenical preparation should never be given if the original dermatitis was vesico-bullous or exfoliative.

UROLOGY.

Wilms Tumour of the Kidney.

F. B. MANDEVILLE AND C. M. NELSON (*The Urologic and Cutaneous Review*, April, 1945) state that recently reported large series of Wilms tumours of the kidney have included very few cases in which a combination of pre-operative radiation and nephrectomy was used. While realizing that a single case can prove nothing, they nevertheless publish a case of five-year survival after such combined therapy. The patient was a girl, aged nine years, with recent pain in the left upper portion of the abdomen. A left-sided retroperitoneal mass was diagnosed and malignant disease (adenomyosarcoma) was proved by aspiration biopsy. X-ray therapy was administered over an eighteen-day period. This was followed by nephrectomy through a large lumbar incision, the important point being early clamping of the renal pedicle to prevent dissemination through the blood stream. No post-operative X-ray therapy was used, and the authors consider this unnecessary unless any suspicious lymph glands or other doubtful tissue is left behind. The post-operative pathological specimen showed marked destruction of cells following a dose of 3,200*r*.

Subcutaneous Urography.

J. H. VASTINE AND M. F. VASTINE (*The Urologic and Cutaneous Review*, April, 1945) state that excretion urography after subcutaneous injection of a diluted solution of a medium usually given intravenously is a satisfactory substitute for the same investigation after intravenous injection. This alternative method is particularly useful in the examination of adults with small veins and of children. A small weal of a 1% solution of "Novocain" is raised near the angle of each scapula and 50 mils of solution are injected in each area. The solution is made up by mixing 20 mils of contrast medium of the type used intravenously with 80 mils of normal saline solution. Films are made at ten minutes' interval after the injection, following the application of a narrow inflated band across the lower part of the abdomen. Fluids are withheld for twelve hours preceding the examination and a dry meal is allowed just beforehand. Intestinal gas formation is cut down by such preparation.

Anuria.

C. P. MATHÉ (*The Urologic and Cutaneous Review*, April, 1945) discusses the differential diagnosis and management of anuria. He accepts the customary categories of pre-natal (circulatory), renal (secretory) and post-renal obstructive types of anuria. Anuria is a potentially or actually shock-like syndrome and calls for accurate diagnosis of the cause in order that effective treatment may be instituted. A careful history taking and complete urological examination help greatly in the differential diagnosis. Repeated determinations of blood pressure, blood volume, blood flow, blood count and nitrogenous waste products are essential in every case. Outside the above three categories there is a post-operative "reflex" type of anuria, which can largely be avoided by anticipation and prophylaxis, for example, intravenous administration of glucose before, during and after operation, gentle handling of tissues, prevention of blood loss, and maintenance of adequate blood pressure by means of ephedrine or "Neosynephrin". When shock is anticipated blood plasma or serum is given throughout and after the operation, and whole blood if much blood loss has occurred. "Carbogen" should be given after all very serious operations for its regulating effect on depressed metabolism. With fluids given intravenously, aqueous adrenal cortex extract is injected as a prophylactic against shock. This hormone controls the permeability of endothelial and tissue cells. The best weapon to combat the secretory type of anuria is an intravenous infusion of glucose or, preferably, sucrose. The most powerful effect is obtained by using either of these substances in a 50% concentration. In this strength 100 mils of glucose are used. Sucrose in this strength is infused in amounts of one mil per pound of body weight. Care is needed with these infused fluids if œdema is present. Fluids should be given by mouth if tolerated, but diuretic drugs are of questionable value. To reduce acidosis, sodium bicarbonate may be given with a 5% solution of glucose by vein. A local method which is often effective in stimulating renal secretion is pelvic lavage with warm sodium bicarbonate solution. Decapsulation is of doubtful value in various nephritic forms of anuria, but some workers still speak highly of it. In the obstructive type of anuria the distal obstruction is relieved by catheterization, otherwise pyelostomy or nephrostomy is indicated. In unilateral obstruction presenting reflex anuria on the opposite side, all the measures necessary to combat secretory anuria are needed as well as removal of the obstruction.

Nephrolithotomy for Recurrent Branching Calculi.

H. G. BUGBEE (*The Journal of Urology*, August, 1944) states that the surgeon may hesitate to perform nephrolithotomy for recurrent branching calculi for three reasons: (i) The possibility of destroying more renal tissue in an already deficient kidney is present. (ii) An extensive parenchymal incision or the freeing of a large calculus may result in post-operative hæmorrhage. (iii) Dense adhesions resulting from the primary operation render dissection difficult and dangerous. He advocates a simplified technique for selected cases. The incision is made below the scar down to the perirenal fascia. The muscular layers are dissected up to the twelfth rib, which is removed. In the cases reported the calculus was then palpable, and an incision was made through perirenal fascia, renal capsule and parenchyma down to the pelvis. The stone was then removed and a nephrostomy drain was placed in the lowest calyx. The author remarks that mattress sutures pass through perirenal fascia, renal capsule and parenchyma, and sufficient traction may then be put on the sutures to control hæmorrhage without danger of tearing through the parenchyma.

Rupture of the Testicle.

ALTHOUGH contusion of the testicle is common, rupture of the *tunica albuginea* rarely occurs. V. S. Counceller and J. H. Pratt (*The Journal of Urology*, September, 1944) record the first case encountered at the Mayo Clinic, and review five others. In spite of severe injury to a sensitive organ, only one-third of the patients suffered severe initial shock. One-half continued strenuous physical activities. The treatment is always surgical, usually orchidectomy. Although the authors were able to conserve the greater part of the testis, they anticipate that atrophy will occur.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 23, 1945, at Lewisham Hospital, Sydney. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff of the hospital.

Congenital Pseudoarthrosis of the Tibia due to Fibrocystic Disease.

DR. L. J. WOODLAND'S first patient was a baby, aged eight months, who had first been examined at the age of two weeks. The mother had noticed that the baby's left foot was deformed. Examination revealed that the left calf was a quarter of an inch less in circumference than the right, and the left lower limb was a quarter of an inch shorter than the right. A moderate degree of left *talipes calcaneus valgus* was present; no other abnormality was detected. When the baby was four months old, the mother noticed that if the left leg was moved the child screamed. There was a history of the baby's having fallen out of a pram a week before. X-ray examination at this stage revealed irregular sclerosis with cystic formation of the middle two-fourths of the tibia. The appearances suggested that there had been an old fracture through the middle third of the tibia, which had united. A recent fracture had occurred through the tibia, just proximal to the united fracture. Proximal to this fracture there appeared to be a large cyst. The fibula was atrophic, and at the distal extremity a fracture was present. The remainder of the skeleton was normal.

The child was treated in a plaster hip spica. X-ray examination three months later revealed slight anterior and medial angulation of the tibia at the site of fracture. There was no evidence of union. The cystic area in the proximal half of the tibia had increased, and another fracture without displacement had occurred through the tibia at the junction of the proximal fourth and distal three-fourths. X-ray examination immediately prior to the meeting, four months after the accident, revealed typical pseudoarthrosis at the site of the fracture, incurred four months previously. The large cyst through which the last fracture had occurred was being obliterated, and appearances suggested that this fracture might unite.

Dr. Woodland said that the interesting features of the case were the evidence of an intrauterine fracture of the tibia which had united, the presence of a fracture at the age of four months, which had led to typical congenital pseudo-arthrosis of the tibia, and finally the occurrence at the age of seven months of a pathological fracture which appeared to be uniting. Whilst there was a history of injury at the age of four months, the X-ray appearances suggested that the fracture which had occurred at that time was pathological. Dr. Woodland considered that this case supported the theory that congenital pseudoarthrosis of the tibia was due to preexisting pathological change, and was not merely an ununited fracture in otherwise normal bone. He pointed out the difficulty in securing union by ordinary bone-grafting methods, and said that he proposed to operate when the child was two years old, and to use double onlay grafts secured from the mother.

Congenital Absence of Femur and Fibula.

Dr. Woodland's second patient was a female, aged five years, who had been born with a short right lower limb. The right foot was at the level of the left knee. The child was first examined at the age of one year. X-ray examination revealed absence of the acetabulum; a small mass of bone about the size of the lower femoral epiphysis was the only evidence of a femur. The fibula was absent. The foot was normal, except that a moderate valgus deformity was present. When the child commenced to walk, she got about well on her right foot and her left knee. At the age of two years she had good balance, and was then fitted with a caliper for her right lower limb. Since then she had moved about freely with a stilt-like extension of the caliper below the right foot.

Hæmangiomata of the Lower Limb.

Dr. Woodland's third patient was a girl, aged seven years. At birth it was noticed that generalized telangiectasis of the whole of the left lower limb was present. There was also a small area of telangiectasis over the left scapula. At birth the left foot was smaller than the right. Since then the whole of the left lower limb had grown more than the right. When the child was first examined at the age of six years, the left lower limb was seven-eighths of an inch longer than the right. The left lower limb was also greater than the right in circumference; the whole of the skin of the limb was speckled with hæmangiomata. There was no elevation of the skin surface, but fine bluish lines traversed the skin, which was more severely affected in the parts not exposed to the sun. The area of hæmangiomata over the left scapula had been cured by a severe sunburn, and was now represented by small, soft scars. X-ray examination revealed no bony abnormality, except an increase in the length of the long bones of the left lower limb.

Dr. Woodland said that the interesting features were the stimulation of bone growth by the increased blood supply, and the cure of one area of telangiectasis by sunburn.

Spondylolisthesis and Hemivertebra.

The last patient shown by Dr. Woodland was a female, aged forty-three years, who gave a history of pain over the mid-line of the back, at about the level of the lumbo-sacral joint, of three years' duration. She said that the pain radiated all over the sacrum and down the lateral aspect of the right thigh to the knee.

On examination, the patient presented the "stove in" appearance of spondylolisthesis. There was no evidence of any root pressure. X-ray examination revealed that the third lumbar vertebra was a hemivertebra, and caused a moderate degree of scoliosis convex to the right. The body of the fifth lumbar vertebra had slipped forward for about two-fifths of the depth of the body. There was evidence of buttressing occurring from the anterior surface of the body of the first sacral vertebra. Dr. Woodland said that, as the patient's general health was not good, for the present it was proposed to treat her conservatively by fitting her with a Taylor's brace. He said that the interesting feature was the association of two different developmental anomalies in the lumbar part of the spine.

Disseminated Sclerosis.

DR. C. M. BURNS showed a young woman, aged twenty-four years. Her illness had begun in 1941 with the sudden onset of acute myalgia, weakness in the limbs and dysuria. Two days later complete paralysis of both legs and retention of urine were present. She then complained of headache and a painful neck. She was admitted to hospital on January 25, 1941. When she was examined then, her temperature was 102° F., the pulse rate was 90 per minute, and the respirations numbered 20 per minute. Moderate neck rigidity with some head retraction and motor paralysis of both lower limbs were present, but sensation was unimpaired. Kernig's sign was not elicited, and the upper limbs appeared normal. The knee jerks, ankle jerks and plantar responses were absent. Retention of urine and constipation were present. Lumbar puncture was performed. The cerebro-spinal fluid was clear and under increased pressure. It contained 95 cells per cubic millimetre, almost all being lymphocytes, and the protein content was 60 milligrammes *per centum* and the chloride content 700 milligrammes *per centum*. The glucose content was not estimated, and attempted culture produced no growth of microorganisms. Treatment was on standard lines. She remained febrile for thirteen days. Four days after her admission to hospital, voluntary movements commenced in the legs, and when a further few days had passed full movements were possible. The reflexes returned, and by February 10, 1941, sixteen days after her admission to hospital, there was no retention of urine. She was discharged from hospital on February 21, 1941.

The patient again presented herself at the hospital on February 23, 1945, four years after the original illness. She said that the bladder and bowel function had not been satisfactory since her illness, and that she now had partial incontinence of urine. She was well otherwise. On examination, the patient was a healthy looking young woman. The full general examination revealed no abnormal features. Examination of the central nervous system revealed that both plantar responses were of the Babinski type. The abdominal reflexes were absent. The knee and ankle jerks were over-active. No other abnormality in the nervous system could be found. The blood did not react to the Wassermann test, and a full blood count showed no evidence of anæmia. The *fundi oculorum* appeared normal. Dr. Burns said that a test meal examination had not been considered necessary, in view of the normal blood findings. The tentative diagnosis of disseminated sclerosis was made on

the evidence of the pyramidal tract lesion. Although an original diagnosis of anterior poliomyelitis had been made in 1941, the present findings suggested that the acute illness then was acute disseminated sclerosis. Some diminution in the incontinence of urine had followed the exhibition of "Prostigmin".

Thyreotoxicosis Treated with Thiouracil.

Dr. Burns's second patient was a girl, aged sixteen years, who had been observed for some years as suffering from congenital heart disease. Four months before the meeting an enthusiastic dentist had extracted twenty teeth under general anæsthesia. The child had failed to make progress after this procedure, and had lost weight consistently. When she was examined two months after the mass dental extraction, the clinical picture was one of thyreotoxicosis, with tachycardia, apprehension, tremor and thyreoid tumour. The basal metabolic rate was +33%. Dr. Burns said that he decided to try thiouracil as a whole treatment. He was influenced in this by the patient's age and by the presence of the heart lesion (a patent interventricular septum). At the time of the meeting the dosage of thiouracil was 0·1 gramme given twice a day. Administration of the drug had been commenced only four days earlier, so no clinical results were yet available.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed or will resume practice as from the dates mentioned:

Dr. D. A. Warden, 152, Ocean Street, Edgecliff (November 12, 1945).
Dr. G. G. L. Stening, 225, Macquarie Street, Sydney (November 27, 1945).
Dr. C. H. W. Lawes, 185, Macquarie Street, Sydney (November 29, 1945).
Dr. R. H. Bettington, 185, Macquarie Street, Sydney (December 4, 1945).
Dr. E. T. Cato, 2, Collins Street, Melbourne (December 4, 1945).
Dr. A. E. Coates, 61, Collins Street, Melbourne (December 10, 1945).
Dr. R. St. J. Honner, 193, Macquarie Street, Sydney (January 7, 1946).
Dr. Harry Green, Q.N. Bank Chambers, Melbourne Street, South Brisbane, Queensland (February, 1946).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on June 13, 1945, DR. H. DOUGLAS STEPHENS, the Acting President, in the chair.

Diaphragmatic Hernia.

DR. MONA BLANCH showed a male patient, aged eighteen years, in whom a diaphragmatic hernia had been successfully repaired by Dr. C. J. O. Brown; at the same time an intractable anæmia which accompanied the hernia had been overcome. Dr. Blanch said that since the boy had been presented to the society on several previous occasions, only a brief *résumé* of the main points of the history was necessary. The main interest of the case resided in the combination of right-sided diaphragmatic hernia and gross anæmia, due either to faulty absorption of iron, or to chronic blood loss, or to a combination of both factors. The patient had first been brought to the Children's Hospital in January, 1930, at the age of three years, suffering from gross secondary anæmia, fever and chest signs thought to be due to a low-grade right basal pneumonia, which an X-ray examination of the chest appeared to confirm. After a period of several weeks in hospital, during which he was given a small blood transfusion, he was discharged to the out-patient department as suffering from von Jaksch's anæmia. For the next twenty-one months he received some form of liver and/or iron therapy more or less regularly, his response to iron being better than that to liver.

In November, 1931, it was decided to present him at a meeting of the society as suffering from obscure anæmia, and an X-ray examination of the chest was made in view of the previous history of lung infection. After viewing the films, Dr. Colin Macdonald suggested the diagnosis of right-sided diaphragmatic hernia, which was subsequently confirmed by a barium meal examination. A large portion of the stomach was found to have passed through the œsophageal opening and to be lying posteriorly in the right pleural cavity. Further X-ray films, in February, 1932, showed that nearly the whole stomach was now in the chest. It was inverted, the greater curvature being higher than the lesser, and the pylorus in the position of the œsophageal opening of the diaphragm. Free hydrochloric acid was present in the stomach contents, which did not contain blood.

In March, 1932, Dr. Charles Osborn attempted to repair the hernia by the thoracic route. Five inches of the seventh rib were resected, and the hernial sac was found to be the size of a large orange. Its contents could be reduced, but would not stay in position. A small opening was made in the sac and peritoneum, and a cannula was tied into the stomach with the idea of deflating it; but the child's condition was not good, so the opening was closed. A month later a second attempt was made, this time by the abdominal route. Owing to recent adhesions there was some difficulty in pulling the stomach through the enlarged œsophageal opening, which was then reduced to normal proportions by the insertion of one mattress suture and one chromicized gut suture after the edges of the opening had been freshened, care being taken not to constrict the œsophagus. The fundus of the stomach was then attached to the under surface of the diaphragm. An X-ray examination ten days later showed that the hernia had recurred. For the next ten years the patient took a variety of different forms of iron more or less regularly; but he was nearly always pale, and often vomiting was troublesome. Occasionally there were periods of good health without iron lasting several weeks; but usually cessation of iron therapy for more than three weeks resulted in a drop of the hæmoglobin value below 40%.

In May, 1941, he was admitted to hospital and given a transfusion of fifteen ounces of blood. In August, 1941, Dr. J. G. Whitaker consented to attempt to repair the hernia by the abdominal route. A test meal examination revealed free hydrochloric acid, but no blood in the gastric contents. The greater part of the stomach was mobilized into the abdominal wound, and though no hernial aperture was felt, it appeared that the stomach was wholly in the abdomen. Ventral fixation sutures were inserted between the stomach and the abdominal wall, and a gastrostomy was performed. The gastrostomy tube came out on the thirteenth day, and a barium meal examination five days later showed the stomach back again in the thoracic cavity.

Since then iron therapy had been continued and the diet had been regulated. On one occasion occult blood was found in the fæces. Early in 1944 Dr. C. J. O. Brown had addressed the society on lung surgery. In view of the success he had attained in this field, he was asked if he would be willing to attempt to repair this boy's hernia at the Alfred Hospital. He did not hold out any great hopes of success, but was willing to attempt it if the boy so desired. As the prospect of a lifetime spent in swallowing iron pills was hardly a cheerful one, the patient was only too glad to accept the chance that was offered; and in June, 1944, just one year prior to the meeting, the operation was performed successfully. Since leaving hospital the patient had had no iron at all, and his colour had remained excellent. Adolescence, which had been much delayed, had progressed rapidly, and now, apart from troublesome acne, he was well. Dr. Blanch said that Dr. Brown had consented to attend the meeting and describe the difficulties he had encountered, and tell how they were overcome.

DR. C. J. O. BROWN said that he was interested to see the original films of the boy's hernia. There were two common types of diaphragmatic hernia. One group included failure of development of the left half of the diaphragm and was principally met with in infants. In these a large proportion of the abdominal contents were in the thoracic cavity, and no sac existed. Such herniæ usually produced respiratory or cardiac symptoms, and operation was necessary to preserve life. The operation was probably best carried out from above. Dr. Brown said that he had had no experience of these herniæ. They were diagnosed by radiological means. The other group included those in which the œsophageal hiatus was enlarged, and in which a sac was present, usually to the left and posteriorly, but occasionally on the right side. They were more frequently met with in elderly people, often

over the age of sixty years. Dr. Brown said that he had operated on seven patients with herniæ of this type; all of them were aged over sixty years. The patient presented by Dr. Blanch was the youngest in this group that he had seen. Patients usually complained of dyspepsia and of recurrent attacks of severe abdominal pain; their condition was usually mistaken for gall-bladder disease. As an example of this type of patient, Dr. Brown recalled an old lady who was thin and miserable and suffered recurring attacks of severe pain, especially upon swallowing. He crushed the phrenic nerve without success. Although Dr. Brown did not think that she was a "good risk", her symptoms became so acute that operation was necessary, and this afforded complete relief. Hernia through the œsophageal hiatus was met with frequently in pregnant women as well as in old age and in conditions giving rise to increased intraabdominal tension. The patency of the foramen was increased in such states.

Dr. Brown went on to say that, reviewing the early films, he would have adopted the course taken by Dr. Charles Osborn and operated through the chest. Usually, however, it was preferable to make the approach from below, because the neck of the sac was more accessible. The operation consisted in reducing the hernia and then removing the sac completely. If this was not done the hernia would recur. In the case under discussion the first mistake was the approach from above, and the second error was in not removing the sac. Non-absorbable sutures were preferable to catgut for repairing the hernia. Strips of *fascia lata* also could be used. Dr. Brown said that he used thread at his operation. When the abdomen was opened adhesions were freed and the neck of the sac was disclosed. Then a large portion of the stomach was reduced. He had then proceeded to free the peritoneum at the neck of the sac, and soon discovered that he had not got the whole stomach out of the sac, for lying in the posterior wall of the sac was a large pouch of stomach, around which he was just able to insert his finger. This was full of gastric content; it was aspirated and reduced. It resembled rather a sliding inguinal hernia, the posterior wall of the sac being the stomach. After this it was a simple matter to sew the sac with thread and close the wound. Some pyrexia occurred during convalescence, which was thought to be due to small residual hæmatomata. Dr. Brown said that his help was enlisted only because the child had passed beyond the age limit prescribed at the Children's Hospital. The operation was facilitated at this stage by the fact that the patient had grown much more than his hernia.

DR. H. SINN said that Dr. Blanch and Dr. Brown were both to be congratulated on presenting such an interesting case and upon having brought it to such a successful conclusion. He commended Dr. Blanch especially for following up the child for such a long number of years, and said that her action was reminiscent of the time when Dr. W. Kent Hughes used to show patients he had kept under observation for twenty years or more. Dr. Sinn said that he admired Dr. Brown's dogmatic utterances on diaphragmatic hernia. He had been confronted with cases among babies and the aged, and in regard to neither could he make up his mind. He remembered about three years earlier showing at a meeting of the society a child, aged twelve months, hoping that the surgeons present might recommend surgical intervention; but this advice unfortunately was not tendered, and no decision was reached. Dr. Sinn said that he had lost sight of the child, but would endeavour to look him up again. Dr. Sinn said that it was refreshing to find someone who took a decided view on these cases, and he thanked Dr. Brown and Dr. Blanch again for the clarity of their views.

DR. R. SOUTHBY said that he was interested in the medical aspects of the problem and especially in the severe accompanying anæmia. Right-sided herniæ were particularly prone to produce a severe type of anæmia. He recalled a case in which a child was subject to so-called acidotic attacks and gradually developed a severe degree of anæmia. Iron was prescribed in large quantities, with improvement of the hæmoglobin value, but another attack would be followed by a severe fall in the level. Dr. R. Allen was examining the child in the casualty department on one occasion and felt something gurgle whilst he was palpating the abdomen. The child's condition immediately improved, and his mother commented that he always improved after she heard such rumbles. On the strength of this, Dr. Allen ordered radiological investigation for diaphragmatic hernia, but this could not be demonstrated. The boy died later, and at autopsy a well-defined hernial sac in the right side of the chest was discovered. Dr. Southby said that anæmia might be due to gastric erosion or to interference with gastric secretion. The latter was probably a greater factor; otherwise melena or hæmatemesis would occur more often. Dr. Southby said that he had recorded the details of this case in THE MEDICAL JOURNAL OF AUSTRALIA, thinking that it was of extreme rarity; but in the next two or three years he was flooded with lists of references of similar cases reported in the American literature, so that the condition was not so unusual as he had at first thought.

DR. BRUCE HALLOWS congratulated Dr. Brown on obtaining such an excellent result. He was interested in the large increase in the percentage of such cases seen in pregnancy. He remembered attending a woman, aged twenty-eight years, who had collapsed after the delivery of her child and nearly succumbed. Two years later she experienced a similar attack, although she was not pregnant, and this proved fatal. He refused certification for her death, and at the coroner's autopsy it was discovered that she had a volvulus of the abdominal contents in a huge left diaphragmatic hernia. This condition might be included as an extra member of the ever-growing list of causes of death from so-called obstetric shock.

DR. KEITH HALLAM said that the condition of partial thoracic stomach resolved itself into two types, that associated with symptoms and that not associated with symptoms. Dr. Hallam was glad to hear Dr. Brown referring to the frequency of its occurrence. He had said at a previous meeting that this condition was so commonly seen radiologically that it was frequently ignored when reports were submitted. In connexion with the type giving rise to symptoms, it was very interesting to note the frequency of pseudo cholecystitic attacks, pseudo *angina pectoris* and persistent dyspepsia. The position in which the patient was examined must be taken into consideration. He recalled examining a patient radiologically two years earlier; no abnormality was detected in the stomach and duodenum, and a cholecystogram revealed some diminished concentration of the dye in the gall-bladder. Later the symptoms changed, and the patient suffered from breathlessness and low substernal pain on bending. Dr. Hallam examined her in the Trendelenburg position and discovered a paraœsophageal hernia, which he was sure had not been there previously. He presumed that the fundus slipped up when she bent over. When a small pouch of stomach which was fixed in the thorax became distended with gas, pain occurred which might simulate angina. Suggestive symptoms such as these should stimulate the medical profession to look for diaphragmatic hernia with greater intensity.

Dr. H. Douglas Stephens said that he had seen this patient on several occasions before, and was impressed by the result. The subject was stimulating. He had operated on several such patients. He had come to the conclusion that in young children, in whom the intestines, part of the stomach and even the spleen might be involved, symptoms were usually met with quite early in life. These usually took the form of pain, vomiting and cyanosis, and signs of dulness and borborygmi were demonstrable in the chest. He had known the chest to be aspirated on the mistaken impression that it contained fluid. Dr. Stephens said that at operation two principal types of the condition were met with. The first was eventration of the left cupola of the diaphragm, which resembled thin parchment, and the intestines forced their way upwards. He felt confident that most of these had a traumatic basis and had their origin at birth. They were best approached by the transthoracic route. The other type was that in which a definite sac was present, the so-called paraœsophageal hernia. The case under discussion was unusual. Dr. Stephens recalled a child in whom a paraœsophageal hernia had been revealed radiologically. At operation it was possible to visualize the intestines being sucked into the thorax. The child died after the operation from intestinal obstruction, and the autopsy revealed congenital atresia of the lower part of the gut. Twelve months later Dr. Stephens was concerned with another child who had a double paraœsophageal hernia. A successful operation was performed on that on the right side, but the child succumbed when the hernia recurred on the other side. Dr. Stephens remembered also another child who was admitted to hospital for a fractured femur. In the course of routine examination the heart was found to be pushed to the right, and X-ray examination revealed a huge diaphragmatic hernia. Operation was urged, but refused. Four years later the child was admitted to hospital in the middle of the night and died from intestinal obstruction. Finally, Dr. Stephens asked Dr. Brown whether crushing of the phrenic nerve had proved to be a useful procedure in his experience.

Dr. Mona Blanch, in reply, thanked the various members for contributing to the discussion. She was interested in

Dr. Southby's remark concerning the large number of cases that had been described in the American literature. The only reference she could find was a case reported by Dr. Stephens in 1932.

Dr. C. J. O. Brown, in reply, said that there had been some confusion and laxity in the terms used to describe the various types of diaphragmatic hernia. It was necessary to be more precise and explicit. It was his belief that every patient with true hernia should be submitted to operation, for obstruction and strangulation were bound to develop sooner or later. However, the œsophageal hiatus hernia of old people could usually be ignored. He did not wish to operate in the case he had described during the discussion. Harrington had reported a series of 120 cases in which he had operated. Most of these herniæ he had repaired successfully. In about 22 instances he crushed the phrenic nerve, or removed a portion of the nerve, or combined both procedures. In a small proportion of these cases this was the only procedure carried out; in others it was used merely as a preliminary measures.

(To be continued.)

The Royal Australasian College of Surgeons.

GORDON CRAIG SCHOLARSHIPS.

THE Council of the Royal Australasian College of Surgeons invites applications for the Gordon Craig Scholarships.

1. These scholarships are awarded for the purpose of furthering post-graduate education in surgery and surgical research.

2. Favourable consideration will be given to applicants who intend to present for a senior surgical qualification.

3. Applicants must have qualified for at least two years.

4. Scholarship holders will be required to carry out whole-time duty at a medical school and/or research institute and/or hospital, although this condition may be varied in exceptional cases.

5. The amount of each scholarship will be at the discretion of the College, but the College is prepared to grant up to £400 a year to suitable applicants.

6. Forms of application, together with details of the regulations governing the award of the scholarships, are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.

7. Applications should be lodged with the Secretary and reach him not later than January 31, 1946.

8. It is proposed to call for further applications at approximately half-yearly intervals and announcements will be made from time to time.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

AN examination for membership of the Royal Australasian College of Physicians will be held in April-May, 1946. The written examination will be held in capital cities where candidates are offering and, for the convenience of candidates serving in the forces, at other centres if the necessary arrangements can be made. A clinical examination will be held in Sydney and also in Melbourne if a sufficient number of candidates offers. Only those candidates whose answers in the written examination have attained a standard satisfactory to the Board of Censors will be allowed to proceed to the clinical examination.

Dates of Examination.—Written examination: Saturday, April 6, 1946. Clinical examination in Melbourne (provisional): Friday and Saturday, April 26 and 27, 1946. Clinical examination in Sydney: Tuesday and Wednesday, April 30, 1946, and May 1, 1946.

Applications.—Applications to appear before the Board of Censors should be made in the prescribed form and must be in the hands of the Honorary Secretary of the College not later than Saturday, March 9, 1946. Candidates should signify in which city they desire to take the written examination and whether they wish to take the clinical examination in Sydney or in Melbourne. Application forms are obtainable from the Honorary Secretary, 145, Macquarie Street, Sydney.

Correspondence.

POVERTY, HOUSING AND HEALTH.

SIR: Surely few will argue against the desirability of reducing poverty and improving housing and health conditions in the community. Dr. E. P. Dark considers that to question the fact of the poverty of the majority of our population shows social ignorance. On that issue some questions seem relevant:

1. Whence come the ever-increasing millions of pounds deposited in the Government Savings Bank?
2. Whence come the many millions of pounds spent annually on liquor, gambling and tobacco?
3. Whence come the huge sums spent at the cinema theatres which are open almost every night in suburb and town?
4. Whence comes the money to pay for the ubiquitous beauty salons which crop up like mushrooms in our midst?
5. Whence come the millions of pounds spent annually on patent medicines?

Much of the complaint of poverty could be traced to wasteful spending on unessentials. The thesis that higher wages and fewer hours of work would produce a healthier populace still remains to be proved.

Yours, etc.,

A. BULTEAU.

Gladesville,
New South Wales,
December 10, 1945.

SIR: May I make a few comments on criticisms by Dr. Dark and Dr. Moore in the journal of December 1, 1945?

Dr. Dark expresses the gratuitous opinion that "only the socially ignorant question the fact of the poverty of the majority of our population". This statement of poverty cannot be accepted as axiomatic, but requires scientific proof. We have already discussed poverty in previous journals. It was pointed out that it is not a simple but a complex state, the result of many factors, for example, low income, laziness, ill health, excessive alcohol consumption, gambling, unnecessary luxuries. The assessment of these factors is a difficult matter, more the work of a statistician or economist than of a medical practitioner. Income is mainly a subject for economists, the basic wage being decided by them, but I may observe that a mere increase in income will not solve poverty. Workers have told me that fifty years ago they lived at least as well on thirty shillings a week as they do now on five pounds a week. Other factors are involved, such as production. About laziness there would not be unanimity in view of Dr. Dark's statement that the "belief that there is a mathematical relationship between the total effective work done and the standard of living suggests an innocently benevolent view of capitalism". In politics I prefer the "golden mean", but consider such a relationship of universal application. The role of excessive alcohol consumption in causing poverty has been stressed by medical writers. Sir Arthur Newsholme stated (1929) that "something like one-fifth of the entire earnings of the wage-earners in Britain is still spent in alcoholic drinks". Incidentally, medical officers of health must have written the equivalent of volumes of facts about defective housing and sanitation in their reports. Gambling and unnecessary luxuries are outside the province of the medical profession, but gambling is an important factor: my gardener mentioned that last year he lost eighty pounds in backing "certs"; he hopes to win it back. Workers have told me that the basic wage is sufficient to provide all necessaries, including friendly society lodge dues, if excessive alcohol, gambling and unnecessary luxuries be avoided. One man told me that he had never earned much more than the basic wage and now owns his home.

The average increase in production per worker in England from 1932 to 1936 was due, not to more work done by the worker, but to the use of more machinery, often necessitated by the go-slow policy.

With reference to the Stockton-on-Tees project, it is not sufficient to mention the number of people involved. The

mortality rates and the probable errors due to the use of small numbers are essential for the comparison, as discussed in chapter 8 of "Frequency Curves and Correlation" by W. Palin Elderton. The object of standardized rates is merely to put the two series on the same basis as regards age distribution when the other conditions have been fulfilled, namely, the series should be large and homogeneous enough and its members should be chosen at random.

With reference to Dr. Moore's statement about the go-slow policy, I have been told by workmen that it is more prevalent in the government service than in private enterprise. I think Dr. Moore overrates the political influence of the medical profession. It has political influence only in so far as it supports the Government's policy.

Yours, etc.,

J. BROWN.

Toowoomba,
January 7, 1946.

TUBERCULOSIS PATIENTS AND HOSPITALS.

SIR: It was announced recently that there is a waiting list of four hundred tubercular patients seeking sanatorium treatment in Victoria. Is there adequate treatment being provided while they wait? Is there an efficient out-patient service where ambulant pneumothorax treatment is provided? I spoke recently to a young soldier being demobilized—he has returned from overseas to find his wife suffering with tuberculosis. There are children. Because of the inadequate sanatorium facilities her doctor has ordered her "rest at home". Pneumothorax treatment has been refused her because of "spread to the other lung". The more expert opinion today is that contralateral spread is an indication, not a contraindication, to collapse therapy. Apparently this patient has no access to more expert opinion, and no treatment is being received of any value. Are others of the four hundred being just left to die?

Yours, etc.,

DAVID B. PITT, M.B., B.S.

12, Montalto Avenue,
Toorak, S.E.2,
Victoria.
December 11, 1945.

THE CENTRAL HOSPITAL, MELBOURNE.

SIR: The old Royal Melbourne Hospital is being reopened as the Central Hospital, and will provide accommodation for patients suffering from venereal diseases, tuberculosis and cancer in special sections, with, in addition, a general section of some 170 beds for both male and female patients with medical and surgical conditions.

The patients in the general section will be served by both full-time resident and part-time paid visiting staff, and applications for appointment to the staff have already been invited.

It is desired to inform members of the Association that the Victorian Branch Council has resolved:

> That, after considering the implications of the system being established at the Central Hospital, and the information promulgated by circular, Council offers to cooperate with the management of the hospital, provided that steps are taken to ensure that all patients of the hospital be referred back to their regular medical attendants on discharge,

and that there is no objection to applications for staff appointments being submitted.

Yours, etc.,

C. H. DICKSON,
Medical Secretary, Victorian Branch of the British Medical Association.

Medical Society Hall,
426, Albert Street,
East Melbourne, C.2.
December 19, 1945.

CAN EPILEPSY BE CURED?

SIR: Periodically in the past, though less in recent years, Dr. Sydney Pern has treated the readers of this journal to a didactic dissertation on focal sepsis. In the issue of December 15 he has further established his claims regarding the cause of epilepsy. Cerebral tissue is subject to the inflammatory reaction of pathogenic bacteria. *Voilà tout!*

Where Muskens is silent and Walter Freeman has little to say, Dr. Pern imputes the "laying down of fibrous tissue on the surface of the brain". Where Lennox and Cobb incline towards a state of cerebral dysrhythmia, Dr. Pern, clinging to his coccal conceit, adumbrates "some intermittent or continuous bacterial entry to the blood stream". Ammon's horn sclerosis he will not have. Nissl need never have existed. The Pacchionian bodies are without mystery. Dandy, Temple Fay, Blackfan, Weed and Penfield are passed by, while Dr. Pern goes back to the gospels, and changing the words "unclean spirit" to "pyogenic cocci", he establishes a pathology of epilepsy as peculiar as that of Paracelsus and as plausible as that of Poseidonios, who, in the fourth century, conceived convulsions as being caused by the nerve centres striving to rid themselves of some *materia peccans* which had got into the blood.

Yours, etc.,

REG. S. ELLERY.

Collins Street,
Melbourne,
December 31, 1945.

Naval, Military and Air Force.

THE TRAINING PRIOR TO DISCHARGE OF CERTAIN SERVICE MEDICAL OFFICERS.

THE Director-General of Medical Services has forwarded for publication the following information on the training prior to discharge of certain service medical officers. A recent Cabinet decision in the matter is as follows.

1. Service medical officers who enlisted within two years of graduation should be granted up to 90 days' leave on full pay of rank immediately before discharge, subject to the production of evidence that an approved full-time course of refresher training is being undertaken; leave granted to be additional to any service leave to which the officer is entitled; salaries received from civil sources during the period of training to be paid to the services.

2. The normal provisions of the Commonwealth Reconstruction Training Scheme in relation to post-discharge refresher training should apply to service medical officers.

There is a further provision that former medical officers whose services were terminated on or after September 1, 1945, and who at the time of termination would have been eligible under the Cabinet decision, may be granted an amount equivalent to 90 days' pay and allowances (less any salary or allowances received from civil sources or from part-time medical employment during period of training) on completion of a course of refresher training approved by the Post-Graduate Committee of the State in which such training is undertaken.

Any such former medical officer who desires to avail himself of this benefit must—

(*a*) on or before 31st January, 1946, obtain the approval of the Post-Graduate Committee (Medicine) of the State in which training was or is being arranged for the course of the refresher training which he has undertaken or proposes to undertake;

(*b*) commence the approved course before 15th February, 1946;

(*c*) complete a course which has been approved by the relevant Post-Graduate Committee;

(*d*) obtain from the D.D.M.S. or A.D.M.S. of any Command or L. of C. Area an application form designed for such purpose.

(*e*) complete the application form and forward it, together with the statutory declaration and certificates mentioned on the form to the D.D.M.S. or A.D.M.S. of the Command or L. of C. Area in which he commenced full-time service.

If the former medical officer is eligible and has duly completed an approved course, the District Finance Officer will make payment to him and allottees at the rate of pay (including allowances) appropriate to the rank held by him immediately before his services terminated, less—

(a) any sum received by the medical officer by way of salary arising from the training, or from part-time medical employment during the period of training;
(b) an appropriate deduction where the medical officer has been supplied with free board and residence during the course or any part thereof;
(c) any sum received by or payable to the medical officer by way of living allowance under the Commonwealth Reconstruction Training Scheme during the course.

Details of refresher training available to meet individual requirements should be sought from the training authority in the State concerned, namely:

Queensland: Secretary, Permanent Post-Graduate Committee (Medicine), University of Queensland, Brisbane.
New South Wales: Secretary, Permanent Post-Graduate Committee (Medicine), 131, Macquarie Street, Sydney.
Victoria: Director, Permanent Post-Graduate Committee (Medicine), Spring Street, Melbourne.
South Australia: Secretary, Permanent Post-Graduate Committee (Medicine), University of Adelaide, Adelaide.
Western Australia: Vice-Chancellor, University of Western Australia, Nedlands.
Tasmania: Vice-Chancellor, University of Tasmania, Hobart.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 234 and 245, of December 6 and 20, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The grant of the acting rank of Group Captain to Temporary Wing Commander E. C. Heffernan (1186) is terminated upon his ceasing to occupy a Group Captain post with effect from 20th September, 1945.

Citizen Air Force: Medical Branch.

The probationary appointments of the following Flight Lieutenants are confirmed: P. R. Bull (257476), J. P. Walsh (257477), W. R. Pitney (257530), J. A. S. Robertson (267525), J. R. Tripp (267568), R. Roxburgh (267591), E. W. Bate (257611), G. E. W. Bennett (267613), D. M. Clarke (257635), R. K. Doig (257656), J. V. Hurley (257659), L. C. Dunlop (267739), L. C. Doubleday (267797), T. Schlight (257727).—(Ex. Min. No. 285—Approved 19th December, 1945.)

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Squadron Leader J. G. Radford (261234), 21st September, 1945, Temporary Flight Lieutenant J. T. Cullen (267165), 25th September, 1945.

The appointment of Temporary Flight Lieutenant J. Tyrer (267564) is terminated on medical grounds with effect from 24th September, 1945.

Reserve: Medical Branch.

Temporary Squadron Leader W. R. F. Fox (263900) is transferred from the Active List with effect from 4th September, 1945.—(Ex. Min. No. 286—Approved 19th December, 1945.)

CASUALTIES.

ACCORDING to the casualty lists received on December 3, 1945, Captain M. J. McNamara, Mosman, New South Wales, and Major J. D. Morris, Oakleigh, Victoria, previously reported "missing, believed deceased", are now reported "died of illness while prisoner of war".

According to the casualty lists received on December 12, 1945, Major W. G. Pickering, Rockdale, New South Wales, is now reported as having been removed from the "seriously ill" list, and Captain D. G. Picone, Cooroy, Queensland, previously a prisoner of war, is now reported "died whilst prisoner of war".

According to the casualty lists received on December 21, 1945, Captain F. A. Cox, Perth, Western Australia, has been placed on the "seriously ill" list, and Captain S. B. McK. White, Neutral Bay, New South Wales, previously stated to be missing, is now reported "missing, believed deceased whilst prisoner of war".

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Moore, David Robert, M.B., B.S., 1945 (Univ. Sydney), Balmain and District Hospital, Balmain.
Morris, Walter John, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Moxham, Ross Moorcroft, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Murphy, John Denis, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Musso, Fedele John Andrew Leo, M.B., B.S., 1945 (Univ. Sydney), Prince Henry Hospital, Little Bay.
Nixon, Eleanor Muriel, M.B., B.S., 1945 (Univ. Sydney), Lewisham Hospital, Lewisham.
Nolan, Marcella Madeleine Therese, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Nowland, Reginald John, M.B., B.S., 1945 (Univ. Sydney), Balmain and District Hospital, Balmain.
O'Connell, Brian Patrick, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
O'Keefe, James Denis Francis, M.B., B.S., 1945 (Univ. Sydney), Brisbane General Hospital, Brisbane, Queensland.
O'Mara, Maxwell Lachlan, M.B., B.S., 1945 (Univ. Sydney), Newcastle Hospital, Newcastle.
Orban, Thomas Desiderius, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Paige, Bruce Flood, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Perry, David Clayton, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Powell, Frederic Arbouin, M.B., B.S., 1945 (Univ. Sydney), Newcastle Hospital, Newcastle.
Puflett, Delmont, M.B., B.S., 1945 (Univ. Sydney), Rachel Forster Hospital for Women and Children, Redfern.
Raftos, John, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Reid, Alison Eva, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Renshaw, Maurice Edward, M.B., B.S., 1945 (Univ. Sydney), Lewisham Hospital, Lewisham.
Rich, David Louis, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Rimmer, William David, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Robertson, Thomas Inglis, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Robey, Ariel Lefliey, M.B., B.S., 1945 (Univ. Sydney), Parramatta District Hospital, Parramatta.
Rothery, Donald Edward, M.B., B.S., 1945 (Univ. Sydney), Lewisham Hospital, Lewisham.
Rothfield, Neville John, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Scott, Dorothy Wolseley, M.B., B.S., 1945 (Univ. Sydney), Balmain and District Hospital, Balmain.
Short, Leslie Frederick, M.B., 1945 (Univ. Sydney), Wollongong District Hospital, Wollongong.
Silvester, George Raymond, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Smith, Warren James, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Smyth, Francis George, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Obituary.

JAMES THOMAS WILSON.

DR. RALPH A. NOBLE, Cambridge, writes: Many graduates of Sydney University who are established in their vocations in various parts of the world will remember the great respect they had for Professor J. T. Wilson. Our medical school owes so much to him, for he was in charge of a most important department from the earliest days through a period of thirty-seven years, until in 1920 he became Professor of Anatomy at Cambridge University.

During his work in Sydney he trained in anatomy many men who became experts in that field and many great clinicians. Perhaps one might mention a few, such as Elliot Smith, A. E. Mills, John Hunter and T. K. Potts. But his influence extended far beyond the school of medicine. For years he was Chairman of the Professorial Board and a member of the senate of the university. Students of all faculties held him in the highest regard. During the Great War he organized the censorship in New South Wales.

Since 1920 at Cambridge he did most important work both for the university and the Empire. He represented the Australian universities on the Executive Council of the Universities' Bureau of the British Empire from 1921 to 1938. He always kept in close contact with Australia and welcomed visitors from overseas at his home in Cambridge.

Since the passing of Mrs. Wilson in November last, the health of Professor Wilson gradually failed, but he maintained a live interest in all scientific matters. He spent a month of this summer at the home of one of his daughters in Scotland, and on his way back to Cambridge visited his birthplace at Moniaive, Dumfriesshire. The end of his life came soon afterwards, on September 2 last. He had achieved much and had interested himself in affairs and in personalities far beyond the science of medicine. At the funeral service held on September 4 it was truly said of him:

> Goodness and mercy all my life
> Shall surely follow me.

His seven children have all been happily established in their various vocations, the three sons serving with the forces now.

WALTER ROBERT GRAHAM.

WE regret to announce the death of Dr. Walter Robert Graham, which occurred on December 31, 1945, at Jersey, Channel Islands.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Williams, John Robert Trevor, M.B., B.S., 1945 (Univ. Sydney), Dubbo Base Hospital, Dubbo.
MacBeth, Robert Duncan, M.B., B.S., 1940 (Univ. Sydney), 13, Iluka Road, Mosman.
Orban, Thomas Desiderius, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.
Clifford, Kevin Patrick, M.B., 1940 (Univ. Sydney), 57, Belmore Road, Randwick.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Boydell, William Herbert, M.B., B.S., 1945 (Univ. Sydney), Western Suburbs Hospital, Croydon.
Church, John Campbell, M.B., B.S., 1941 (Univ. Sydney), 10, Toongarah Road, North Sydney.
Collins, Ian Stuart, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Dunn, Eric Robert, M.B., 1945 (Univ. Sydney), Wollongong District Hospital, Wollongong.
Gailey, William George, M.B., B.S., 1938 (Univ. Sydney), Stratton Hall, 26, Simpson Street, Bondi.
Graham, David Lindsay, M.B., B.S., 1940 (Univ. Sydney), 16, Edward Street, Gordon.
Roger, John Robert Osborne, M.B., B.S., 1939 (Univ. Sydney), 110, Manning Street, Taree.
Stephenson, Noel Lavendar, M.B., B.S., 1943 (Univ. Sydney), c.o. 93, Bathurst Street, Sydney.
Brennan, Desmond James, M.B., 1939 (Univ. Sydney), 25, Park Road, Burwood.
Campbell, William Hay, M.B., B.S., 1938 (Univ. Sydney), 3, Bertha Road, Cremorne.
Dowling, John Laidley, M.B., B.S., 1937 (Univ. Sydney), 1, Rose Bay Avenue, Bellevue Hill.
Griffiths, William James, M.B., B.S., 1942 (Univ. Sydney), Queanbeyan.
Lebanon, Miriam, M.B., B.S., 1944 (Univ. Queensland), 5/124, Francis Street, Bondi.
Rundle, Herbert Wesley, M.B., B.S., 1945 (Univ. Sydney), The Maitland Hospital, Maitland.
Spence, Olaf McClure, M.B., 1936 (Univ. Sydney), (NX77361 Captain), 102, Northwood Road, Lane Cove.

Books Received.

"The Osseous System: A Handbook of Roentgen Diagnosis", by Vincent W. Archer, M.D.; 1945. Chicago: The Year Book Publishers Incorporated. 8" × 5¼", pp. 320, with many illustrations. Price: $5.50. Australian, 40s.

"Pulmonary Tuberculosis in the Adult: Its Fundamental Aspects", by Max Pinner, M.D.; 1945. Springfield, Illinois: Charles C. Thomas. 8¾" × 5¾", pp. 592, with many illustrations. Price: $7.50.

"Psychology in General Practice", edited by Alan Moncrieff, M.D., F.R.C.P.; 1945. London: Eyre and Spottiswoode Limited. 8½" × 5½", pp. 199. Price: 12s. 6d.

Diary for the Month.

JAN. 17.—Victorian Branch, B.M.A.: Executive Meeting.
JAN. 22.—New South Wales Branch, B.M.A.: Council Meeting.
JAN. 23.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 24.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 25.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 31.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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